T H E S I S:

B R O N C H I E C T A S I S:-

An Enquiry into some points of its Etiology, Morbid Anatomy, Pathogenesis, Treatment, and the value of X'Rays in diagnosis,

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Introductory:-

Of all the diseases which medical men are called upon to treat Bronchiectasis seems to be one of the most difficult. Fortunately it is a comparatively rare disease but the lives of those whom it attacks are rendered a burden to themselves and to all around them. Socially such cases are shunned and in most instances they are unable to continue their employment on account of the horrible foetor which they exhale. Added to this there is the mental effect on the patient. In a very short time he becomes of a profound melancholic disposition, shuns everyone, and soon life becomes almost intolerable. For some time my attention has been called to these cases and during my studies at the Brompton Hospital for Diseases of the Chest, London, I have had special opportunities of trying various remedies in their treatment, through the kindness of the Physicians there. I have had placed at my disposal the clinical and also the post-mortem records of the hospital and together with the cases I have had under my charge I have had good material to go on in making a study of this disease. I am indebted to Drs. Powell, Mitchell, Bruce, Williams, Kingston-Fowler, Kidd, Acland and Mackenzie for permission to use the records of the cases under their charge.

In studying these cases my investigations have been along the following lines.

Apart from treatment, I have paid special attention to an enquiry into the mode of production of Bronchiectasis with special reference as to whether Pleural adherency is one of the main factors in its production. In this connection it has been necessary to enquire minutely into the etiology and morbid anatomy of this condition and I record these results for their own value as well as for their bearing on this enquiry. The result of my experience has led me not to rely entirely on clinical data alone, as I consider that any such investigation based upon that only is almost worthless and consequently in each case I have endeavoured to present a complete clinical and pathological picture. I have gone over the records of 3,227 post-mortems done at the hospital here, and amongst them I found 72 cases of Bronchiectasis, and it is on these cases combined with a review of the cases in the clinical records, which do not come to the post-mortem table, the cases which came under my charge in the hospital, and those which were attending the out-patient department, that the results of my investigations depend. A list of the literature on this subject, which I consulted, and

which has given me much valuable help I give at the end of this paper. The section on surgical treatment* is my part of the work of a paper undertaken in conjunction with Dr. Acland, Physician to St.Thomas's Hospital.

The Photographs illustrating my paper which were taken at the Royal College of Surgeons,London, through the kindness of Professor Stewart, comprise all the more important specimens in the museums of the Brompton Hospital for Diseases of the Chest, St. Thomas's Hospital, Royal College of Surgeons, and also one specimen from the Victoria Park Hospital for Diseases of the Chest, London. I have to record my indebtness to the physicians at these various hospitals for permission to photograph and study these various specimens. A study of the clinical cases in hospital here under the X Rays has also afforded me much valuable help.

The microscopic work was done at the University College laboratory.

Etiology:-

Frequency:-

I find Bronchiectasis occurring in 72 cases out of 3,227 post-mortems (chest disease) made at this hospital - that is equivalent to 2·2%.

These statistics agree with those of Biermer who records 2% out of 400 post-mortems; while Williglk records 8% out of 4,517. It must however be borne in mind in drawing any conclusions from the above statistics that they are based on post-mortem records. The vast majority of the cases which come into hospital here leave never to return and consequently never come to the post-mortem table; but the same may be said of cases of other diseases. I endeavoured to compare the number of cases of Bronchiectasis with those of other chest diseases found in the clinical records, but I gave it up as useless because experience taught me that several cases of so-called Bronchiectasis - diagnosed clinically - when they came to the post-mortem table were not cases of Bronchiectasis at all; and also that a number of cases in which no evidence of it existed during life, when they were examined post-mortem proved to be undoubted cases of Bronchiectasis - these were invariably associated with tuberculosis. Therefore to have based statistics on the clinical records alone,

* Has been omitted from this paper/

would have been worthless and the conclusions deduced from them fallacious.

Throughout my paper I have made use of the term "Pure Bronchiectasis" to denote those cases in which dilatation of the tubes were found not associated with Tuberculosis; the cases which occurred as a result of Aortic Aneurism,* Mediastinal tumour, foreign bodies and Syphilitic stricture I have termed "Traumatic Bronchiectasis."

* when the term "Aneurism is mentioned, it refers throughout to "Aortic" unless otherwise defined

Age:- The following are the ages (at death) in 64 cases of Bronchiectasis.

5 - 10 years of age	3·2 %
10 — 20 years " "	20·3 %
20 — 30 years " "	25· %
30 - 40 years " "	28·1 %
40 - 50 years " "	14· %
50 - 60 years " "	6·2 %
60 — 70 years " "	1·6 %
70 — 80 years " "	1·6 %

The above results confirm the statement of Lebert and Biermer, that the disease arises most frequently in "early and middle adult life", i.e., 53.1% of my cases occurring between the ages of 20 and 40 years. These statistics deal with the ages at death and as we know that the disease may last for long periods, it gives us little data to go upon in estimating the actual time of onset. It is worthy of note that the two cases over 60 years of age were associated with chronic Pulmonary Tuberculosis and were very marked types.

It is of further interest to compare the ages of the different pathological types thus:-

Pure Bronchiectasis - ages (at death) of 40 cases:-

5 — 10 years of age	5· %
10 — 20 years " "	25 %
20 — 30 years " "	15 %
30 — 40 years " "	34· %
40 — 50 years " "	10 %
50 — 60 years " "	8 %

Tubercular Bronchiectasis:- ages (at death) of 22 cases:-

Between 10 and 20 years.	13·6%
" 20 and 30 years	41·%
" 30 and 40 years	18·2%
" 40 and 50 years	22·7%
" 50 and 60 years	4·5%

Traumatic Bronchiectasis:- ages (at death) of 8 cases - (included in the above 40 Pure cases);-

Between 10 and 20 years.	38%
Between 20 and 30 years.	12%.
Between 30 and 40 years	25%.
Between 50 and 60 years	25% } all cases of aneurism

The above tables point out the interesting fact that although most of the Pure cases of Bronchiectasis occur between the ages of 30 and 40 years, those associated with tuberculosis tend to occur chiefly between 20 and 30 years; evidently taking on the tubercular type of onset, as evidenced by the fact of 41% of cases occurring between those years. Another point is brought out and that is, that a marked proportion of the tubercular as compared to the Pure cases occur between 40 and 50 years of age (22·7%). I take this to indicate that these cases may have originated during the period of liability to phthisis (20 - 25 years) but that fibrosis which is a marked associate of these tubercular Bronchiectasis cases, gives chronicity to a certain percentage.

Two cases occurred between the ages of 70 and 80 years, one of general Bronchiectasis and the other of cylindrical Bronchiectasis, and both shewed evidence of Pulmonary tuberculosis. The figures dealing with traumatic Bronchiectasis shew that those cases in association with aneurism occurred between 50 and 60 years and those due to foreign bodies between 10 and 20 years of age.

Sex:-

In 70 cases of Bronchiectasis I find the following:-

	No. of cases	males	females	
Pure Bronchiectasis	36	26	10	males = 44%
Traumatic Bronchiectasis	9	9	0	females = 23%
Tubercular Bronchiectasis	25	19	6	
Total cases:	70	54	16	

According to these statistics we find that there is a marked liability of the male to attack and this is what most others shew. We find that in tubercular Bronchiectasis alone there is also a marked liability to attack in the male and the significance of this is more clearly shown when we compare the liability of the male to attack from Pulmonary tuberculosis. Osler states that in Pulmonary tuberculosis the female is slightly more liable to attack, if anything, than the male, and the Registrars-General's statistics on Pulmonary tuberculosis shew that the death-rate from that disease from 25 to 35 years is slightly greater in the female - the opposite of that in Bronchiectasis.

In the traumatic cases it is worthy of note that all occurred in males.

Hereditary phthisical history:-

It is of interest to enquire what percentage of cases of tubercular Bronchiectasis give a hereditary history of phthisis in comparison with those of Pure Bronchiectasis. I find that in 22 cases of tubercular Bronchiectasis there is a phthisical history (Parents, brothers and sisters), in 41% and in 36 cases of Pure Bronchiectasis (including the traumatic cases) 14% only. As regards Pulmonary tuberculosis 384 cases analysed by Wilson Fox gave a hereditary phthisical history in parents and collaterals in 48%: 12,146 cases by Squire in grandparents, parents and collaterals in 62.34%, and 1,000 analysed by Cotton in parents and collaterals 36%. (Diseases of Lungs and Pleura, Wilson Fox. page 529). Thus it will be seen that the percentage of cases of tubercular Bronchiectasis giving a hereditary history of phthisis corresponds fairly accurately with that in cases of Pulmonary tuberculosis.

However seeing that 14% of cases of Pure Bronchiectasis give such a history it would be misleading to put much stress on this point as a factor in diagnosing as to whether a case of Bronchiectasis was tubercular or not, apart from Bacteriological evidence.

Mode of Onset:-

It is at all times difficult to get a high degree of accuracy on this subject owing to patients being in some instances unable to recall the exact onset of their disease. Therefore statistics to be at all reliable, must be based on exact information. In the following statistics, the onset has been determined from the time of the first appearance of a continuous cough originating either from Bronchitis, Pleurisy, Pneumonia or combinations of these. I have tabulated the mode of onset of the different types as I think it helps to lucidate the subject and render it more valuable.

Mode of onset of 64 cases of Bronchiectasis:-

Pure Bronchiectasis - 34 cases:-

Chronic Bronchitis	80 %
Pneumonia (lobar)	14 %
Pleurisy	3 %
Pleuro - Pneumonia	3 %

Tubercular Bronchiectasis - 21 cases:-

Chronic Bronchitis	76 %
Pleurisy	14 %
Pneumonia (lobar)	10 %

Traumatic Bronchiectasis - 9 cases:-

Chronic Bronchitis	56 %
Pleurisy	33 %
Pleuro - Pneumonia	11 %

Bronchiectasis: all 64 cases:-

mode of onset:-	no. of cases	%
Chronic Bronchitis	48	75%
Pneumonia (lobar).	4	11.5%
Pleurisy.	4	11%
Pleuro-Pneumonia.	2	3%

It will be seen from the above statistics that the great majority of cases have a chronic Bronchitic onset. I mean by this, starting with a cough which has become continuous, in most cases getting gradually worse; sometimes with recurrent acute exacerbations. Lebert states that ¼ of his cases gave a previous history of Emphysema, another ¼, a history of Pleurisy or Pneumonia and in a large number, a history of long-continued Bronchitis.

My own experience is that in most cases it is an absolute impossibility to get from a patient whether he has suffered from Emphysema or not. I admit Emphysema is commonly associated with chronic Bronchitis but to record Emphysema as an onset in such cases is to me extremely fallacious. In no case in hospital have I ever got from the patient a history of Emphysema as an independent onset of his disease. In my cases the onset from Pleurisy and Pneumonia coincides with that of Lebert and I consider that 25% of all cases represents fairly accurately the onset from these diseases. Some writers state that Pneumonia (lobar) is a much more frequent mode of onset. Cases however come up to hospital giving a history of Pneumonia as the onset of their disease, but on strict enquiry it is found that the disease had existed in a slight form for some time previous to that, but that an attack of Broncho-pneumonia or even Septic Broncho-pneumonia had rendered the latent disease more evident, and led the patient to look upon it as the onset of his disease. Such cases, I've no doubt are recorded as having originated in Pneumonia (lobar).

The mode of onset in Traumatic cases is fairly definite. The cases associated with Aneurism began with cough, pain in the side and dyspnoea later.

Those associated with Mediastinal tumour all began with a dry hacking cough, slight frothy mucoid expectoration and dyspnoea gradually increasing, out of all proportion to the physical signs. Pleurisy intervened in several cases at a later date. In all the cases associated with foreign bodies, Pleurisy was the mode of onset. Such a Pleurisy is usually recurrent or leads on to Empyema. Hence in children where any history is given as to the "swallowing" of a foreign body, if the case commences with Pleurisy especially if it is recurrent or leads on to Empyema, we should strongly suspect the presence of a foreign body in the bronchus and have the chest carefully X rayed. (See British Medical Journal, April 12th. 1902, page 894).

Associated diseases:-

The diseases in association with Bronchiectasis are so various that no definite statement can be made of much practical value. Many of them appear as coincident factors and have little effect on the course of the disease. Several, however, occurring after the developement of the Bronchiectasis have marked effects in influencing its course; the diseases bringing about these results being Pleurisy and Empyema, Broncho-pneumonia, Septic Broncho-pneumonia and Gangrene. The occurrence of the two latter bringing about in most instances a fatal issue.

The following are the most important diseases which occurred in association with my cases:-

Pure Bronchiectasis:-

Occurring before the developement of the Bronchiectasis:-

(in order of their frequency):-

1. Measles and Whooping Cough. 2. Influenza. 3. Syphilis. 4. Smallpox. 5. Rheumatic Fever (one case). 6. Ague (one case).

Occurring after the developement of the Bronchiectasis.

(In order of their frequency):-

1. Broncho-Pneumonia. 2. Pleurisy. 3. Septic Broncho-Pneumonia. 4. Empyema.

Tubercular Bronchiectasis:-
(All occurring after the onset of the disease).
(In their order of frequency):-
1. "Influenza". 2. Pleurisy. 3. "Congestion of the Lungs". 4. Pneumothorax. 5. Empyema.

Traumatic Bronchiectasis:-
All occurring after the development of the disease.
(In order of their frequency):-
1. Pleurisy. 2. Empyema. 3. Pulmonary tuberculosis (one case).

It will be seen from the above, that in the cases of Pure Bronchiectasis, Whooping Cough, and Influenza are found most frequently in association with them. It is generally acknowledged that Whooping Cough and its sequelae are the starting point of many cases of Bronchiectasis and also that some of the worst types develop after its occurrence. However, too much stress ought not to be put upon this, because in all diseases it is common to get a history of Whooping Cough. Influenza undoubtedly plays a much more important part in the development of Bronchiectasis than has hitherto been assigned to it, both in the tubercular and pure types. Of course it would be erroneous to accept all the statements of patients with regard to these so-called attacks of "Influenza", as being true Influenza. Nevertheless one is inclined to agree with the statement made by Osler, "That one of the most distressing sequels of Influenzal Bronchitis is the development of diffuse Bronchiectasis." (Principle of Medicine: Osler, 4th. edition: page 94.).

Broncho-pneumonia, Septic Broncho-pneumonia and Gangrene occurring in the course of the disease undoubtedly may have marked effects; the first by causing its spread, the second by shortening its course and in many instances leading to a fatal result, and the third in increasing the gravity of the case by causing great destruction of lung tissue and in some cases leading the production of pneumothorax and subsequent death.

In the Traumatic cases the great and constant associates are Pleurisy and Empyema; these being most commonly found in cases of foreign bodies and mediastinal tumours.

Symptoms:-
It is beyond the scope of this paper to deal in detail with the various symptoms of Bronchiectasis, and as nearly all are agreed as to these, it would be

useless to do so. But as the statistics of my cases on Haemoptysis are of some interest, I record them.

Bronchiectasis: 62 cases:-

Percentage of cases in which moderate, profuse ~~and~~ or fatal haemoptysis occurred:-

	no. of cases.	males	females	males females
Pure Bronchiectasis	33	46%	45%	45·5%
Tubercular Bronchiectasis	22	56%	33%	50%.
Traumatic Bronchiectasis	4	29%	no females	29%

To explain the above, when I record that 46% of males had profuse, moderate ~~and~~ or fatal haemoptysis, I mean 46% of male cases only and the same applies to the females.

I find in the 62 cases above mentioned that fatal, profuse ~~and~~ or moderate haemoptysis occurred in 45.1%. We find Walsh recording that he has not observed haemoptysis except in cases associated with mitral stenosis or with tubercle. Biermer records its occurrence in 1/8 and Lebert in 1/6 of their cases. Wilson Fox states that it is not rare, and though probably most common in the presence of tubercle, it may occur and prove fatal without this complication (Diseases of Lungs & Pleurae, page 122). Thus it will be seen that my statistics record a much higher percentage of cases of haemoptysis than most of the others, but I can vouch for the accuracy of the figures, as in most cases not only is the haemoptysis recorded but its amount. It will be noticed that the tubercular cases show a slightly greater liability to fatal, profuse and moderate haemoptysis. It is worthy of note that out of the 62 cases, 3 had fatal haemoptysis, all occurring in cases of pure Bronchiectasis, two from the rupture of a pulmonary aneurism into a dilated tube, and one from extensive ulceration of lung tissue implicating a branch of the Pulmonary artery.

In the cases of traumatic Bronchiectasis profuse haemoptysis occurred in 2 cases or 29%, and of these one occurred in a case of aneurism with associated Pulmonary tuberculosis, the other in a case of mediastinal tumour with extensive ulceration of lung tissue.

Morbid Anatomy.

It is not my object in treating of the morbid anatomy of Bronchiectasis to give a detailed account of all the various morbid processes; this can be found in any pathological work on the subject. One of the main objects of this paper and to which I hope nearly all else has contributed has been an enquiry into the important and difficult question as to the part played by Pleural adhesions in the production of Bronchiectasis. Some hold (as Biermer) that changes in the pleura play an important part in causing the dilatation of the tubes, others (as Kingston Fowler) hold the opposite view. Hence I have dealt only with those morbid processes which I considered had a special bearing on that question and which have been my chief study.

Since the time of Laennec there has been no change in the description of the various dilatations as cylindrical, fusiform, and saccular. As regards the Moniliform variety, I met with it rarely and it is not of much pathological interest.

In describing the various cases I have adopted a clinical and pathological classification and make use of it, only because it has afforded me the most convenient and accurate method of classifying and describing my cases, so as to bring out the various points which are of special interest.

Classification:-

I. Bronchiolectasis
- Acute
- Chronic.

II. Bronchiectasis
- A. Pure
  - 1. Chronic Bronchitic
  - 2. Broncho-Pneumonic
  - 3. Chronic Pneumonic (or Fibrotic)
  - 4. Pneumonic
  - 5. Pleuritic.
- B. Tubercular
- C. Traumatic.
  - 1. Aneurism (Aortic).
  - 2. Mediastinal tumour.
  - 3. Foreign Body
  - 4. Syphilis.

Bronchiolectasis:-

This form is described as occurring in an acute form in children, but it may also occur in a chronic form in adults especially in old subjects. Photograph No. (IX.) shews a typical specimen of the acute type. Through the kindness of Dr. Acland, Physician to St. Thomas's Hospital, I am able to add a microphotograph of this specimen, shewing the Bronchiolar dilatation, acute peribronchitis and associated collapse. Amongst my own cases I find one example of the chronic type (see morbid anatomy types, case A.M. No. 40.). This case occurred in a female 70 years of age with a history of winter cough for several years, but no acute illness. The condition was associated with tubercular ulceration of the larynx, but no trace of tubercle was found in the lungs on microscopic examination. It is worthy of note that the condition was associated with a high degree of Emphysema, with bullae occurring along the edges of the lungs. With the exception of the emphysema the lung tissue was more or less in a natural state, but the left lung shewed some consolidation anteriorly with very slight fibrosis. The right pleura was free except for a slight adhesion at the extreme apex, and over the left lung generally were a few scattered adhesions. The bronchioles were generally dilated throughout both lungs, forming small cavities about the size of peas, filled with muco-pus.

Bronchiectasis:-

The various types which I have adopted in my classification have the following characteristics:-

Chronic Bronchitic type (see Morbid anatomy, Cases Nos. 1-6)

This type had a history of having originated with cough which became chronic. The varieties of dilatation usually found were slight general cylindrical, combined with slight Saccular, or slight general cylindrical alone. The lung tissue was usually slightly firmer than normal with some degree of emphysema. The Pleurae were usually very slightly generally adherent. It must be noted however that Pleurisy seemed to intervene in some cases to almost mask the original type as in Case H.W. No. (5.), where owing to attacks of Pleurisy

(double) the Pleurae had become densely adherent and the lung tissue more indurated. Attacks of Broncho-pneumonia may mask the condition so as to render it almost impossible to determine the original type.

Broncho-Pneumonic type:- (see morbid anatomy. Nos. 12-24).

This type formed a large percentage of my cases. Their exact pathological onset was difficult to determine and in many cases impossible to find out. Some undoubtedly originated from Broncho-pneumonia in childhood usually occurring after measles and whooping-cough. The majority gave as their onset a history of cough which became chronic. Most of the cases seemed to have originated in adult life as the chronic Bronchitic types did, but Broncho-pneumonia supervening in one or both lungs seemed to have caused a partial or general spread of the disease; But at whatever stage this condition presented itself post-mortem, it was distinctive. Broncho-pneumonia chiefly occurs in young subjects, but I think there is little doubt that it occurs more frequently in the adult, in a more or less subacute or chronic form than is generally supposed.

The dilatation occurring in these types varied, i.e. cylindrical, general cylindrical, saccular, cylindrical and saccular combined, and slight cylindrical with large gangrenous cavities. When cylindrical and saccular were combined, the saccular variety largely predominated. Ulcerations of the Bronchi were very rarely seen, and occurred only in the saccular type.

The lung tissue presented variations, i.e. regular or irregular patchy consolidation, slight fibrosis, dense fibrosis, and scattered pigmented fibroid nodules. Occasionally slight emphysema or a spongy condition of the lung were found; also localized areas of collapse or lobar collapse especially of lower lobes (pleuritic types). If recent septic Broncho-pneumonia had occurred the minute Bronchi and Bronchioles were filled with purulent material.

Tubercular Bronchiectasis:- (See Morbid Anatomy, Cases 45-40)

It appeared as if all these cases were primarily tubercular, starting with a cough which became

continuous. The onset of some of these cases was in Pleurisy and very rarely in Pneumonia (tubercular?)

The Bronchial dilatations most commonly occurred in the upper lobe, though in some cases they were general. The variety which occurred by far the most frequently was cylindrical. General cylindrical, saccular, saccular and cylindrical combined were found in a few cases. Ulceration occurred in one case only, but it is stated by some authorities that ulceration is most common in the presence of tubercle. The condition of the lung ~~condition~~ tissue varied. It may be stated that generally the most extreme cases of dilatation occurred in densely fibrotic lungs, but also in lungs with marked tubercular consolidation (caseous and fibro-caseous) with but slight fibrosis. The Pleurae were adherent over those parts of the lung where the dilatations existed, in most instances and were generally dense and thick. It is also to be noted however that adhesions occasionally were present over parts of the lung in which no dilatation existed.

Chronic Pneumonic or chronic fibrotic types:-
(See Morbid Anatomy Cases Nos. 9-11)

This condition may arise independent of an attack of acute lobar pneumonia. The onset in all cases was cough which became continuous and got gradually worse. There was no history of any acute illness. The condition was in every case unilateral, but the other lung was occasionally slightly affected, due to a Broncho-pneumonic spread from the affected lung.

The variety of dilatation usually associated with this condition was 'marked' saccular combined with slight cylindrical'. A single large saccular dilatation alone was found in one case.

The lung tissue was uniformly and densely fibrotic in the affected lung, while the opposite lung was usually congested and emphysematous. Where Broncho-pneumonic spread had occurred to the unaffected lung, the lung tissue there shewed scattered areas of fibroid induration.

The Pleura was markedly adherent and thickened over the affected lung and where Broncho-pneumonic spread had occurred to the opposite lung, the pleura, at least, was usually somewhat adherent over those parts where the slight dilatations existed.

Pleuritic Types:- (See Morbid Anatomy Cases No. 35)

I have only one case to record of this type and it is bilateral, occurring after a double pleurisy with effusion.

The dilatations were of the saccular variety and were limited to the lower lobe in one lung, but occurred in both lobes in the other lung, where the lung tissue was generally collapsed and fibrotic.

The Pleurae over both lungs were free, with the exception of some recent adhesions over their posterior surfaces.

Pneumonic Types:- (See Cases: Morbid Anatomy, Nos 28-34)

These cases all began with acute lobar pneumonia.

The varieties of dilatation found were saccular, cylindrical, general cylindrical, saccular and slight cylindrical combined. If the dilatation was general throughout the lung, it was of the cylindrical variety; if limited to the lower lobe, it was usually of the marked saccular variety. The lung tissue associated with the dilatations in every case was densely fibrotic.

The Pleura in every case was at least adherent over the sites of the dilatations. In some cases the opposite lung had become slightly affected owing to Broncho-pneumonic spread.

Traumatic Bronchiectasis:- (See Morbid Anatomy, Cases Nos. 36 & 44).

These cases usually began with cough which became continuous, in some cases with Pleurisy, which was sometimes followed by Empyema.

The dilatations were either cylindrical, cylindrical combined with saccular or fusiform.

The lung tissue around the dilatations was either emphysematous, or densely fibrotic, or collapsed.

The Pleurae as a rule were adherent over the sites of the dilatations unless Pleural effusion or Empyema were present.

It should be mentioned that the dilatations in these cases were either the results of pressure on the bronchi from Aneurism, or mediastinal tumour, or were due to the presence of foreign bodies in the bronchial tubes, or narrowing of the Bronchi from syphilitic scarring and subsequent bronchial stenosis.

Types of Bronchiectasis:-

It is of interest to determine the frequency of the various types of Bronchiectasis and in order to put them in as concise and lucid a manner as possible I arrange them in tabular form.

Frequency of the various types, in 70 cases:-

Pure Bronchiectasis: (including Traumatic types) :- 66 %.

Tubercular Bronchiectasis :- 34 %.

Traumatic Bronchiectasis: (includes in above Pure types) :- 13 % of all cases.

It will thus be seen that tubercular Bronchiectasis forms a fairly large proportion of all the cases. Biermer found only 3 cases of tubercle associated with bronchial dilatation present in all the cases analysed by him.

Trojanowski however found tubercle in 21 of his 68 cases of Bronchiectasis.

Wilson Fox (Diseases of Lungs and Pleurae. page 117.) makes the statement "that taking all cases of Bronchiectasis collectively, tubercle will be found to be a common complication."

Extent of the Bronchial dilatations:-

Unilateral or Bilateral affection:-

In 69 cases I find the following:-

	Bilateral	Unilateral
Pure Bronchiectasis (including Traumatic types)	67%	33%
Tubercular Bronchiectasis	54%	46%
In all 69 cases	62·3%	37·7%

The marked preponderance of Bilateral cases is worthy of note. Lebert in 54 post-mortems found a bilateral affection in 48% and a unilateral affection in 52% (nearly). Wilson Fox (Diseases of Lungs and Pleurae Page 114) states "that the affection is limited to one lung in about half the cases.

Traumatic Bronchiectasis (included in the above Pure types).

In 9 cases:-

	cases	%
Bilateral	2	22%
Unilateral.	7	78%

Thus in these cases unilateral affection predominated largely. It is worthy of note that of the two cases presenting Bilateral affection, one was in a case of Aneurism which pressed on the Right and left Bronchus and the other was in a case of foreign body in one bronchus with associated tuberculosis in the other lung.

Situations of the Bronchial dilatations in the lung:-

Pure Bronchiectasis (including Traumatic types):-

A. In 30 Cases of Bilateral affection:-

all lobes affected in one lung.	48%
upper lobe alone " " " "	2%
middle lobe alone " " " "	0%
Lower lobe alone " " " "	15%
upper + middle lobes alone " " " "	0%
upper + lower lobes alone " " " "	5%
middle + lower lobes alone " " " "	0%.

B. In 15 Cases of Unilateral affection:-

Right- lung was affected in 13 cases.	86·6 %
Left- lung. " " " 2 cases	13·4 %.

all lobes affected in	60%
upper lobe alone "	13%
middle lobe alone "	0%
Lower lobe alone "	4%.
upper + lower lobes alone "	20%.

Traumatic Bronchiectasis:- (included in the above 15 Pure types.)

In 7 cases of Unilateral affection:-

Right- lung was affected in: 6 Cases	86%
Left- lung " " " : 1 case	14%.

all lobes affected in	86 %
upper lobe alone in	14 %
middle lobe alone in	0 %
Lower lobe alone in	0 %

Tubercular Bronchiectasis:-

A. In 13 cases of Bilateral affection:-

all lobes affected in one lung in	62 %
upper lobe alone " " " " "	24 %
middle lobe alone " " " " "	0 %
Lower lobe alone " " " " "	4 %
upper + middle lobes alone " " " "	4 %
upper + lower lobes alone " " " "	0 %
middle + lower lobes alone " " " "	0 %

B. In 11 cases of Unilateral affection.

	%
Right lung affected in 8 cases =	72.7 %
Left lung affected in 3 cases	27.3 %

	%
all lobes affected in	0 %
upper lobe alone " in	18 %
middle lobe alone " in	0 %
Lower lobe alone " in	55 %
upper + middle lobes alone " in	9 %
upper + lower lobes alone " in	18 %

I add Lebert's statistics on these points for comparison (quoted by Wilson Fox, Diseases of Lungs, page 114).

In 28 Cases of Unilateral Bronchiectasis:-

	Cases	%
all lobes affected in	12	42%
upper lobe alone " "	6	21%
middle lobe alone " "	1	3%
middle + lower lobes alone "	9	32%

The following points of interest are brought out in the above statistics of my cases. In unilateral cases of Bronchiectasis of all types, there is a much more frequent affection of the Right lung as compared with the left. Wilson Fox (Diseases of Lungs, page 114) makes the statement that the lungs suffer in about equal frequency. My statistics disagree with that statement.

In the cases of Pure Bronchiectasis, both unilateral and bilateral, there is a marked preponderance of affection of all the lobes in one lung. Lebert's statistics on this point are not of much value, seeing he has not differentiated his cases. The small percentage in which the upper lobe alone was affected in both bilateral and unilateral cases and the absence of affection of the middle lobe are noteworthy features.

Dealing with bilateral cases of tubercular Bronchiectasis, the affection of all the lobes in one lung (62%) is very marked, whereas in the unilateral cases there are no examples of this. Again, the marked preponderance of affection of the lower lobe alone in unilateral cases (55%) stands out in marked contrast to that in bilateral cases (7%)

My cases also shew that the great frequency of affection of the upper lobe in tubercular Bronchiectasis as compared with Pure Bronchiectasis is not so marked as one is led to believe.

Associated Pathology:-

The lesions in association with Bronchiectasis are very varied.

Dealing first with Heart lesions, I find them associated with Bronchiectasis in 4%. (3 cases), of my 72 cases; all being lesions of the aortic valve, i.e., stenosis, regurgitation, stenosis and regurgitation combined. Two occurred in cases of Pure Bronchiectasis and one in a case of tubercular Bronchiectasis. It is worthy of note that the condition was associated with an extremely fibrotic condition of the lungs.

Pericarditis with effusion occurred in 5% of 70 of my cases, all in Pure Bronchiectasis. Pericardial adhesions occurred in 18% of the 72 cases, due to an extension of the pleuro-fibrotic process around.

Cerebral abscess was found in 11.1% (8 cases) of my 72 cases, all occurring in cases of pure Bronchiectasis. It is of importance to note that four of these followed surgical operations, i.e., two for Empyema in association with Bronchiectasis and two for drainage of Bronchiectatic cavities. One of the cerebral abscesses, apart from the four above mentioned, was found in a case which had undergone treatment by subcutaneous injections of Formalin, and occurred in the right frontal lobe, giving rise to practically no definite symptoms or physical signs. Apart from those associated with Empyema, these abscesses were found in cases with large sacculated putrid cavities.

Their situations are given below:-

Situations of the 8 cerebral abscesses:-

(1) Left temporo-sphenoidal lobe.
(2) Left motor area (upper part.)
(3) Right occipital lobe.
(4) Right Frontal lobe.
(5) Right Facial motor area.
(6) Midway between Fissures of Rolando and Sylvius (right.)
(7) Cerebellum (middle lobe.)
(8) One not defined.

It is also of interest to note the tubercular lesions other than those in the lungs associated with the cases of tubercular Bronchiectasis, especially with regard to the occurrence of tubercular Laryngitis and Enteritis.

In 21 cases of Tubercular Bronchiectasis in which the condition of the Larynx and Intestines were carefully noted I find the following:-

Tubercular Laryngitis	39%
Tubercular Enteritis	14%
Tubercular Laryngitis + Enteritis (not including any of the above)	28%
Cases presenting none of the above	19%

It will thus be seen that 81% of these cases had either Tubercular Laryngitis, Enteritis or a combination of these - a very high percentage indeed. On looking into the statistics of these affections in cases of Pulmonary Tuberculosis in the post-mortem records at the Brompton Hospital, I find that in 565 consecutive cases 42.8% had associated Tubercular Laryngitis and 67% associated Tubercular Enteritis.

Hypertrophic-osteo-pulmonary arthropathy occurred in several of the cases, but as no special note was made on this condition, in many of the cases, I can make no definite statistical statement.

Lardaceous disease of the kidneys and liver was found in several cases, more frequently indeed than one finds in association with cases of Pulmonary Tuberculosis.

The rarer complications included Pneumothorax, one case (Tubercular) and complete transposition of the abdominal and thoracic viscera (in a case of general cylindrical Bronchiectasis).

Prognosis:

In dealing with prognosis, it is convenient first to get as definite an idea as possible as to the duration of such cases, and also as to the main factors which influence the course of the disease. In endeavouring to obtain accurate information as to the duration, one is presented with many difficulties, the chief being that of determining the exact time of onset. I have, nevertheless, been able to ascertain this with as high degree of accuracy as is possible. The duration has been estimated from the onset, which I have defined as the time of the first appearance of a continuous cough, whether starting with Bronchitis, Pleurisy, Pneumonia or combinations of these.

The following are the statistics as to the duration of Bronchiectasis in 62 of my cases:-

Pure Bronchiectasis: 41 cases:-

Duration:-

up to 1 year	1 to 2 years	2 to 3 years	3 to 4 years	4 to 5 years	5 to 6 years	6 to 10 years	upwards of 10 years
26·8%	12·3%	14·4%	9·8%	4·8%	2·4%	14·6%	12·2%

Tubercular Bronchiectasis: 21 cases.

Duration:-

up to 1 year	1 to 2 years	2-3 years	3-4 years	4-5 years	5-6 years	6-10 years	upwards of 10 years
28·4%	33·3%	4·8%	0%	0%	4·8%	9·5%	19·2%

61·4%. 0%.

Traumatic Bronchiectasis: 9 cases: (included in the above 41 Pure cases):-

up to 1 year	1 to 2 years	2 to 3 years	3 to 4 years	9 to 10 years
2 cases	2 cases	2 cases	2 cases	1 case

88·8%

All 62 cases of Bronchiectasis:-

up to 1 year	1 to 2 years	2 to 3 years	3 to 4 years	4 to 5 years	5 to 6 years	6 to 10 years	upwards of 10 years
27·4%	19·3%	11·2%	6·4%	3·2%	3·2%	14·8%	14·5%

Lebert's statistics: in 52 cases:-
Period of survival was:-

Of one year in	21·1%
Of 1 to 2 years in	4·4%
Of 3 to 5 years in	30·7%
Of 6 to 10 years in	15·5%
Of upwards of 10 years in	25·0%

With regard to the duration of cases of Bronchiectasis one usually fails to find in the text-books any definite statement, but if it is made, it is usually to the effect that no reliance can be put on statistics dealing with this subject, owing to the difficulty in determining the exact time of onset, and also because of the great apparent variations in their duration. I admit that there is some difficulty in determining the exact onset in some cases, but with care, in the vast majority; one can fix it with much certainty; also the fact that a disease

presents great variations in its duration, is not to be taken as proof that it cannot be accurately determined.

For comparison I have put along side of my own statistics those of Lebert.

Dealing with my own statistics it will be seen that in all cases of Bronchiectasis of whatever type, the number of cases having a duration of 1 year and under amounts to 27.4%. Those having a duration of from 1 to 2 years amount to 19.3%. Thus 46.7% died at or within 2 years and 57.9% at or within 3 years of their onset. 9.6% of the cases had a duration of 3 to 5 years, 14.8%, a duration of 6 to 10 years, and 14.5% a duration of 10 years and upwards.

Comparing these statistics with Lebert's, one may say that they agree (generally) in the percentage of cases having a duration of 1 year. They also agree in the number of cases having a duration of from 6 to 10 years, otherwise they are at variance. It is of interest to note that in my cases of Pure Bronchiectasis 39.1% died within 2 years, and 53.8% within 3 years of their onset. In comparing these with the Tubercular Bronchiectasis, one finds in the latter that 61.7% died within 2 years of their onset, and this is what one would expect, seeing that in all such cases the Tubercular lesion is the predominant factor in the disease. One has also to note that in all cases of Bronchiectasis 9.6% have a duration of from 3 to 5 years; on the other hand in Tubercular Bronchiectasis no cases have a duration within those years, but that 19.2% have a duration of 10 years and upwards. This is a point of some interest for us to consider. In enquiring into those Tubercular cases having a duration of over 5 years, one finds that the time of onset in all, was either at about 15 to 16 years of age, or in later adult life about 40 years of age. The period of onset in those cases having a short duration was usually between 20 and 30 years of age. Thus one would gather from this, that the development of Tubercular Bronchiectasis at an early age and also in later adult life lends chronicity to such cases, and may lead one to a more hopeful prognosis. But this is really of little practical importance when one takes into account that many cases of Tubercular Bronchiectasis are not diagnosed during life. Notwithstanding one should bear this point in mind when one meets with those cases.

Another point I would mention in this connection and it is this, that I am strongly of opinion that many of those cases of Tubercular Bronchiectasis, which develop in early and in late adult life are primarily bronchiectatic and that the Tubercular

affection is secondary. I advance this opinion, not only on statistical but on pathological grounds. I admit that the vast majority of cases of Tubercular Bronchiectasis are primarily Tubercular and agree with the general opinion that Pulmonary Tuberculosis as secondary to it is rare; but it does occur. This point is worthy of further research. In this connection I might also mention that both the cases of Bronchiectasis I record over 60 years of age had associated Pulmonary Tuberculosis, which was undoubtedly secondary.

It will be seen that many of the cases have a prolonged course, even as long as from 30 to 40 years; those cases of Tubercular Bronchiectasis having a duration of 10 years and upwards shewing 19.2% as compared with those of all cases of Bronchiectasis shewing 14.5%, confirming the view that Bronchiectasis adds chronicity to many cases of Pulmonary tuberculosis.

In the cases of traumatic Bronchiectasis one finds that they all died at or within 4 years of their onset except in one, which was associated with Aortic Aneurism; also, that all the cases associated with foreign bodies in the Bronchi, died within 2 years of their onset.

Haemoptysis:-

It is of interest to enquire as to what effect the occurrence of haemoptysis has on the prognosis. As I record the statistics on haemoptysis later I will merely mention the facts deduced from them. I find that in 62 cases of all Bronchiectases 45.1% had a history of moderate, profuse or fatal haemoptysis. Fatal haemoptysis alone occurred in 4.8% or in 3 cases.

It is of further interest to record the statistics of cases in which haemoptysis occurred thus.

The duration of each case is given under the extent of the haemoptysis which occurred, to show the influence (if any) on the duration.

Pure Bronchiectasis: in 40 cases:-

no haemoptysis	Streaking	moderate Haemoptysis (3ss to 3vi.	Profuse Haemoptysis (½ pint to 1 quart)	Fatal Haemoptysis.
15 months	4 months	12 months	9 weeks	4 months
15 months	11 months	12 months	6 months	over 1 year
1 year	1 year	14 months	10 months	9 years
3 years	2 years	3 years /	1 year	one (2) /
4 years	2½ years		2½ years	
4 years	3 years		4 years	
5 years	3 years		10 years	
7 years	4 years.		10 years	
10 years /	5 years		about 20 years	
	about 6 years		about 30 years /	
	8 years			
	about 10 years			
	about 25 years			
	about 30 years /			

Tubercular Bronchiectasis: 21 cases:-

no haemoptysis	Streaking	moderate Haemoptysis	Profuse Haemoptysis	Fatal Haemoptysis
9 months	8 months	11 months	13 months	0 /
9 months	13 months	12 months	14 months	
8 years	1¾ years	3 years	1¾ years	
several years /	2 years	12 years	2 years	
	10½ years /	about 20 years /	6 years	
			8 years	
			14 years /	

Haemoptysis does not seem to influence the course of the disease in any marked degree; in fact it cannot be said that the duration of those cases in which profuse haemoptysis occurred is much less than those in which there were streaking or no haemoptysis.

As mentioned above, Fatal Haemoptysis occurred in 3 cases and all were in cases of Pure Bronchiectasis; two being the result of the rupture of a pulmonary aneurism into a dilated tube, and one the result of extensive ulceration of lung tissue implicating a branch of the pulmonary artery.

Other factors influencing the prognosis:-

Broncho-pneumonia:

Attacks of Broncho-pneumonia especially septic Broncho-pneumonia occurred in a great many cases, in most leading to a partial or extensive spread of the disease, and in some bringing about a fatal issue. The occurrence of septic Broncho-Pneumonia should be looked upon with grave suspicion in well-marked cases of Bronchiectasis.

Ulceration in and around the dilated tubes occurred rarely but if extensive may lead to gangrene and death.

Gangrene occurred in several of the cases and had a marked effect in shortening their duration. Some cases are recorded by others where Gangrene occurring near the surface of the lung led to the production of pneumothorax and subsequent death, but in none of my cases did this occur. Photograph No. (XII.), however shews a large gangrenous cavity about to burst through the Pleura.

Cases having a chronic Bronchitic origin undoubtedly give a more hopeful prognosis and this is well shewn in my cases of this type (see Morbid anatomy Cases Nos. 1-6 & others.)

It is stated that cases originating in Pneumonia (lobar) and Pleurisy run a shorter course, but although this is true of some cases, no definite statement can really be made. I tabulate below the statistics on this point, as being of some interest.

Duration of cases originating in Pneumonia (lobar) and in Pleurisy:-

Pure Bronchiectasis: 6 cases:

	originating in Pneumonia (lobar)	originating in Pleurisy	
duration →	9 weeks 10 months 11 months 7 years 8 years.	4 years.	

Tubercular Bronchiectasis: 3 cases:

	originating in Pneumonia	originating in Pleurisy	
	13 months.	12 months 1¾ years.	

Unilateral or Bilateral affection:-

One finds the statement generally made that cases of Bilateral affection present a worse prognosis. I think if one takes this to mean cases originating bilaterally it may be fairly accurate. However on examining my cases I find that in Pure Bronchiectasis those which presented Bilateral lesions post-mortem had an average duration by no means shorter than those presenting unilateral lesions. In fact I would state that the average duration of the Bilateral cases was somewhat longer.

I have been unable to ascertain what proportion of these Bilateral cases originated as unilateral affections, but the vast majority of them had bilateral lesions on admission to hospital. Some of these bilateral cases had a duration of from 20 to 30 years; but it is difficult to state accurately the date of affection in either lung on pathological evidence alone. Nevertheless, studying this point both from clinical and pathological grounds I am strongly of opinion that many of these cases undoubtedly originated as bilateral affections; in this I am supported by views expressed here.

It is of interest to note that most cases of bilateral affection having a long duration, were cases of general cylindrical Bronchiectasis of the smaller tubes, with slight uniform fibrosis of the lung tissue, having a chronic Bronchitic onset.

Again when one enquires into unilateral and bilateral lesions in cases of Tubercular Bronchiectasis one forms an entirely different opinion. Those cases which presented bilateral lesions had undoubtedly a much shorter duration.

Various statements are made as to the prognosis in cases associated with heart disease; the general opinion being that it shortens their duration. Amongst my 72 cases of Bronchiectasis I find only 3 cases with associated heart disease. All these were Aortic lesions and occurred in cases of Pure Bronchiectasis. The duration of these 3 cases was 1 year, 3 years and about 20 years respectively. One often finds it stated in the textbooks, that heart disease in association with Pulmonary Tuberculosis gives a more favourable prognosis, but my experience goes to convince me that this is a fallacy. I can therefore only make a general statement and say, that there is no evidence to shew that heart disease complicating cases of Bronchiectasis leads one to give a more unfavourable prognosis. But I must mention that such cases undoubtedly have a greater tendency to profuse Haemoptysis.

Septicaemia and pyaemia:-

These are grave complications in Bronchiectasis and nearly always lead to a fatal issue. In three of my cases they were the immediate cause of death, and in two of these Pericarditis was found in association. The duration of those cases in which Septicaemia and Pyaemia occurred was 1½years, 2 years and 7 years respectively.

Cerebral abscess:

Occurring in the course of Bronchiectasis is always fatal. It occurred in 11.2% (8 cases) of my 72 cases. Therefore when the symptoms and signs of cerebral abscess develop a fatal result may be looked for.

Amyloid disease of the Kidneys:

The general opinion is that such a complication adds to the gravity of the case. I have been unable to enquire into this point fully, but the majority of the cases in which it was found post-mortem had a short duration. However I can make no definite statement on this point.

The question may be asked Do we ever get a "cure" in any cases of Bronchiectasis? One is led to believe that undoubtedly such a result happens in early life in cases of acute Bronchiectasis, but although this may be true, there is no direct evidence for this assertion. Nevertheless the probability is that it does happen and it is quite reasonable to suppose that this occurs in young subjects where the tissues are actively growing, and where as a result of lung disease following measles, whooping-cough, etc., the tubes return to their normal condition again after the subsidence of the acute disease. But this does not happen in every case, because either owing to the lowered vitality of the tissues or the virulence of the morbid process, cough is kept up and the dilatation progresses and becomes permanent: then I doubt whether a so-called "cure" ever occurs.

In the present state of our knowledge the best result we can hope for in most cases, is a comparative arrest of the destructive processes, and there is no doubt that this can be secured in some cases. The cry of the present age is all for "cures", but like Pulmonary Tuberculosis we should have at our command two factors which are almost allied to cure, and these are, Prevention and Early Diagnosis. One has only to see the results of effective treatment in early cases to acknowledge the inestimable boon of early diagnosis.

Pathogenesis:

It is not my object here to discuss all the many and varied theories as to the mode of production of Bronchiectasis. In the light of all these theories it has been my endeavour to study the part played by pleural adhesions in causing bronchial dilatation. The method I have pursued in this enquiry, apart from the literature on the subject, has been to analize in detail all my cases as to the morbid anatomy found associated with this condition, combined with their clinical history. I have had many opportunities here of examining the post-mortem specimens microscopically and have taken occasion to observe in a series of cases under the X Rays the conditions presented to us in early and late stages of the disease. Thus I here present my own views on this subject, and at the same time take the liberty of criticising some opinions held by various authorities, in the hope that it may make my contentions clearer.

The three great factors which are concerned in this enquiry are the Bronchi, Lung tissue, Pleura, and Respiratory mechanism. In a healthy subject there is a delicate balance between all these, and consequently morbid processes which affect any one of them must necessarily affect this balance. Of all the structures which maintain this respiratory balance we may say that the lung tissue is the weakest, and under certain abnormal conditions the most likely to give way first, to any continued extra pressure put upon them. Thus in emphysema, where the chief disease is primarily in the bronchial tubes we have the alveoli yielding to increased pressure. In some such conditions we have the bronchi dilatating, and why this should occur is the object of our enquiry. There are two factors which must be explained and these are; what circumstances bring about the initial dilatation, and this having occurred, what renders it permanent? Whatever the explanation is, given the requisite amount of weakening of the bronchial wall (Bronchitis), there must be a cause for its dilatation. There must either be brought to bear on the walls of the weakened tubes an extra continued pressure, a great diminution of the surrounding lung resistance, or a combination of these two in variable degrees.

Let us see how these factors come about, to produce dilatation of the Bronchial and alveolar systems, by an enquiry into the various morbid changes in these structures which maintain the respiratory balance.

We may have morbid changes in the:-

(1) Respiratory muscles (Inspiratory.
(Expiratory.

(2) Lung tissue and Pleurae.

(3) Bronchi and Bronchioles.

(4) Blood vessels and Lymphatics.

(5) Lymphatic glands.

Morbid changes in the Blood-vessels and alterations in the Blood supply:

Primary morbid conditions in the Bronchial and Pulmonary arteries are rare, and as such cannot be a factor of any great importance. It is very rarely indeed that we find an atheromatous condition of these vessels alone, and if it does occur, in most cases it is part of a general condition, which affects all the structures equally, and hence the respiratory balance is uniformly affected.

Changes in the lymphatic vessels alone are usually part of a general condition and as such may be dismissed.

Morbid changes in the Respiratory mechanism alone:

These may be either part of a general or a local condition.

The local condition only we are concerned with here.

Increased Inspiratory Effort:-

Inspiratory effort may be increased or diminished. The factors which bring about an Increased Inspiratory effort are acute disease in the bronchi, bronchioles or alveoli, i.e., acute bronchitis, capillary bronchitis, Broncho-pneumonia, etc. These, however, are only temporary conditions as acute processes, and as such as far as Inspiration is concerned are of subsidiary importance. But are there any conditions which lead to a continuous increase of Inspiratory effort? The only condition I can find of this is adherent Pleura over fairly normal lung, because in most

cases of adherent Pleura with underlying lung disease, there is decreased Inspiratory effort. As the former condition is rare it cannot be of any great importance alone as a common factor in the production of Bronchial dilatation.

Decreased Inspiratory effort:-

This may be brought about by disease of the Pleura, i.e., acute Pleurisy or disease of the alveoli i.e., Emphysema, chronic Pulmonary tuberculosis, chronic Pneumonia and other fibrotic processes. The first of these, acute Pleurisy leading on to effusion may become a continuous factor, altering the Respiratory balance and as such demands attention; but I will deal with this later on. But we may also have a chronic Pleuritis giving rise to adherency of the Pleura.

Let us now examine these two factors which decrease Inspiratory effort:

(1) Chronic Pleuritis with adherency.

(2) Chronic disease of the alveoli.

Chronic Pleuritis with adherency with no associated morbid lung condition:

It may be said that there is no evidence that such a condition exists unless in primary or secondary chronic affection of the Pleura (cancer, etc)., and some other such conditions in which Bronchiectasis is never found.

Nevertheless I firmly believe that it may occur as a simple chronic Pleuritis and probably more frequently than we are led to suppose, in association with chronic disease of the bronchi (pleuro-bronchitis) What would be the effects of such a condition? The results of the continued cough, with closed glottis would be to lead to an increased air pressure (expiratory) in the tubes and alveoli. One would expect that if this pressure were continued long enough that the weakest part of the Respiratory tract would yield and give rise to Emphysema or in extreme cases Bronchiolectasis.

But we know that Emphysema is very rarely associated with Pleural adhesions, though it is a very common associate of chronic Bronchitis. Thus we must look to some other terminal affection as the result

of this process namely Bronchiolectasis (chronic). This is just what we find occurring in some cases, and we do get such a condition as this in old subjects and I record such a case in my statistics (see Morbid anatomy cases A. M. No. (40)). I admit that this is a comparatively rare condition as far as post-mortem evidence goes, as the combination of the factors bringing it about so seldom occurs, but that it does occur I have no doubt. Therefore bearing in mind the associated bronchial disease and the fact that the lung tissue is healthy or almost so, the factor which apparently plays an important part as to whether Emphysema or Bronchiolectasis is to be the result is adherency of the Pleura. It is a very doubtful point whether we can really get this latter condition apart from adherency of the Pleura, personally I don't think we can.

Decreased Inspiratory effort associated with chronic alveolar disease i.e.

Chronic Pulmonary tuberculosis or chronic fibrotic diseases, etc., with or without Pleural adherency:

This condition may be a general or local one, but in all we have associated chronic bronchial disease as well.

Let us consider the general condition with Pleural adherency, where the whole lung is thus affected.

The conditions we have presented here are a diminished Inspiratory, with an increased expiratory effort, associated with closed glottis. One would expect that the results of this would be a uniform dilatation of all the tubes especially the larger. How is this then brought about? The alveoli are practically thrown out of action in such a condition, but even if they are patent they are not prone to dilatation by the extra expiratory pressure, being well supported by the existing consolidation or fibrotic conditions. The adherency of the Pleura to the chest wall and also the adherency of the lobes to one another play their part during forced expiration with closed glottis in presenting alveolar dilatation. Hence by these factors associated with closed glottis, during increased expiratory effort, the diaphragm being fixed and the abdominal muscles brought into play, the tendency is all towards the extra-air pressure being exerted on the larger tubes. We must also take into account that in all these chronic processes, the larger bronchial tubes are relatively more weakened by the prolonged bronchial catarrh, and the pressure outside the whole

length of the Respiratory tract being comparatively uniform, owing to the greater surface they present to the air pressure they are more prone to dilatation. The result of this is a tendency to a general dilatation of all the larger tubes and hence we get a general cylindrical Bronchiectasis. The effect of the diminished Inspiratory effort also plays a minor part in the subsequent dilatation of the tubes by proportionately diminishing the air pressure on the alveoli during inspiration, and also by diminishing the uniform pressure outside the tubes owing to the imperfect inflation of the surrounding alveoli. It also acts by causing a gradual contraction of the alveoli and may eventually lead to their obliteration with increasing fibrosis.

But it may be said that we get the above general condition without Pleural adherency in a few cases associated with Bronchiectasis. I hold that the grounds for this assertion are very doubtful. In all my cases I can't find one example of this. Kingston Fowler (Fowler and Godlee, Diseases of Lungs, page (130))., states that Pleural adhesions are sometimes absent from such conditions and hence are not a factor of much importance in the production of Bronchiectasis. He founds the above assertion on the fact that in some cases of Bronchiectases which were operated on at the Brompton Hospital here to effect the drainage of Bronchiectatic cavities, no pleural adhesions were found. On discussing this subject with Dr. Fowler, he informs me that these cases were disgnosed clinically as having pleural adhesions over the bronchiectatic cavities, and therefore clinically no reliance can be put on statements as to pleural adherency occurring in these cases. However it must be borne in mind that these operations were done in cases where medical treatment had failed and were undertaken as a last resource in virulent cases, usually presenting large cavities, many of them being partly gangrenous. Now one finds that the large single (very rare) or large multiple cavities occur in cases of chronic pneumonia or such other allied conditions and are in many cases of a gangrenous saccular type. It is also noteworthy that the tubes themselves in association with these cavities are in many cases but slightly dilated, and the cavitation may have come about chiefly by a slow gangrenous process of the tubes supervening upon an ulcerative one. Such cases undoubtedly occur with little or no adherency, but they are rare. To some extent the absence of dilatation of the tubes themselves associated with those large cavities can be accounted for by the fact, that the extra-air pressure during increased expiratory effort is taken off them by the presence of these cavities and also because of the absence of Pleural

adherency. It is a notable fact that one finds the association of large cavities with marked dilatation of the tubes communicating with them in cases of Pleural adherency. It is worthy of mention that all the cases here which were operated on and which subsequently came to the post-mortem table, shewed marked pleural adherency, and it must be remembered in this connection, that most of these cases which were surgically treated, died not long after the operation. In searching the clinical records here I failed to find one case of Bronchiectasis treated surgically which long survived the operation. I will return to the part played by pleural adherency in all these cases later.

Morbid local conditions, i.e., chronic pulmonary disease with Pleural adherency.

It is a well-known fact that such conditions are frequently found associated with Bronchiectasis. The effects produced by these conditions are much the same as in the previous general condition above described, only limited to one part of the lung.

The air-pressure is equal throughout the bronchi and bronchioles, and the weakest parts of the respiratory tract in such cases are the terminal bronchi and bronchioles, owing to the inflammatory process there. The effect of acute capillary bronchitis or Bronchopneumonia-for it is difficult to dissociate these two conditions - is in most cases to lead to collapse of the tributary areas of these bronchioles. These areas I believe, may remain collapsed and become fibroid and many such areas may become so affected. Hence we get as a result of this a disturbance in the respiratory balance, and the terminal tubes, being necessarily the weaker through the acute and subsequent chronic inflammatory processes, and also by the constant soaking in the retained secretions, must necessarily be more prone to dilatation during increased expiratory effort with closed glottis. During forced expiratory effort with closed glottis, the diaphragm being fixed, the force of the abdominal muscles exerted in an upward direction on the diaphragm, the apex of the lung being practically fixed, tends to lessen the longitudinal diameter of the lung and increase the lateral diameter, especially that of lower part of the lung. This leads to the air-pressure being expended on the lateral and peripheral parts of the lung especially over the lower portions and hence in the above conditions where the terminal bronchi are specially weakened, the tendency is towards a terminal dilatation and consequent production of saccular Bronchiectasis.

I hold that if the Pleura is adherent, this tendency to saccular dilatation may be lessened, but if it does occur with Pleural adherency the associated cylindrical dilatations are much more marked than they otherwise would have been.

The frequency with which Pleural adherency is associated with chronic Broncho-pneumonic processes is very difficult to determine, but I am strongly of opinion that it occurs more frequently than we are at present led to believe. Anyway, all my cases which originated from Broncho-pneumo had associated Pleural adherency post-mortem. But this is of little or no importance in this connection, seeing it is impossible in these cases to determine whether or not it existed "ab initio".

It is of interest to note here, that in cases of Bronchiectasis, the result of mediastinal tumour, where the main bronchu was pressed on, that the most marked dilatations occurred in the lower lobe, shewing the greater tendency of this lobe to undergo dilatation under equal conditions. Also it must be borne in mind that Bronchiectasis is sometimes associated with practically normal surrounding lung tissue.

Changes in the Bronchi and Bronchioles alone:

Bronchi alone:

Chronic Inflammation of the bronchi may exist for long periods without producing any marked change in the surrounding lung tissue. A Peri-bronchitis usually results, but in time a chronic interstitial process may be set up in the lung tissue around and lead to some slight general fibrosis. In most cases there is no associated Pleurisy. The effects of this continued cough with closed glottis is to lead to the air-pressure being chiefly expended on the terminal respiratory tract, i.e., the alveoli, and hence may lead to emphysema. Here there is no associated Pleural adherency. In some few cases the bronchioles may suffer as well as the alveoli under certain conditions, leading to a general Bronchiolectasis and when such a condition is presented post-mortem there is slight general pleural adherency. The significance of this latter condition is well worthy of note.

Bronchioles and alveoli alone:

(1) Acute capillary bronchitis.
(2) Broncho-Pneumonia.
(3) Pneumonia (lobar);

these may be either general or local in their distribution.

All cases of acute capillary Bronchitis and Broncho-pneumonia are not by any means followed by Bronchiectasis, but that some cases are, is an acknowledged fact. How this may come about I have already endeavoured to show. There is one point however which I don't feel clear about. The above conditions by a plugging of the bronchioles may lead to collapse of their tributary areas and subsequent fibrosis of these, if the condition becomes chronic.

However, the collapse may not be permanent, and the alveoli may be subject to re-inflation, under forced expiratory effort with closed glottis. Is it possible that the re-inflation of such an area may lead to the production of an internal emphysematous bulla which may be the origin of our saccular Bronchiectasis, or Bronchiolectasis with their initial thin walls? Or is it that the saccular Bronchiectasis has its origin in the dilated terminal bronchus or bronchiole? Wilson Fox (Diseases of Lungs and Pleura, page 119), states "that the walls of dilated bronchi are in some cases found thinner and in others thickened, is a mere accidental circumstance depending on the relations between the degrees of stretching and of inflammatory thickening which they have undergone." But in this connection it must be remembered, that we get small terminal saccular dilatations with thin walls, associated with cylindrical dilatation of the same tube which has its walls there comparatively distinctly thicker than the former, which admits of no such explanation as that advanced by Wilson Fox. I consider that there is a much deeper reason for this condition, and I hold it is to be found in some such explanation as I have given above.

We know that Bronchiectasis may arise as a sequel to unresolved Lobar Pneumonia and by some the latter is looked upon as a fairly frequent cause. Biermer states that 1/4 of his cases of Bronchiectasis gave a history of having started in acute Pneumonia. The accurate determination as to whether these so-called attacks of acute Pneumonia are the originators of the Bronchiectasis is a matter of the greatest difficulty. Hence I would be inclined to put little value on Biermer's statement. That it does happen seems certain but I believe it to be a rare occurrence (see Ewart on Bronchiectasis, Allbutt's system of medicine page 64).

Changes in the Pleura alone:

(1) Acute Pleurisy with effusion.
(2) Plastic Pleurisy.

That Bronchiectasis may follow upon Pleurisy with effusion is a well-known fact. The variety of dilatation

which is most frequently found in these cases is saccular, and the lung tissue is usually in an intensely fibrotic condition. Owing to the collapse of the lung tissue all the pulmonary structures are in such a weakened condition as to allow of their dilatation under increased expiratory effort with closed glottis. If the alveoli are not permanently collapsed - as I believe is not commonly the case - one would expect that they would be the first to undergo dilation and so lead to the condition of Emphysema, but this is not so. Is it that the alveolar structure and terminal bronchi have been practically obliterated as does occur in some of these cases? What part Pleural adherency plays here I am unable to determine. In such cases, however, that dilatation of the tubes can occur without it, seems highly probable.

Morbid changes in the Lymphatic glands at the root of the lung: *

The enlargement of these glands was very marked in all my cases, most of them being tough and indurated and a few calcareous. One is apt to overlook the part these may play in such conditions.

In chronic Pulmonary Tuberculosis we find a similar condition of these glands, in all probability a secondary infection from the diseased lung tissue (this is even denied by some authorities). In some cases of Broncho-Pneumonia in children which have ended fatally I have noticed a high degree of enlargement and induration of these glands. In cases of Bronchiectasis might one not reasonably suppose that such a condition of glandular enlargement exists prior to the dilatation of the tubes. If this is the case, what would be the effect on the Pleura specially. Apart from any slight pressure effects on the root vessels and lymphatics, these enlarged glands must lead to the production of a certain degree of stagnation in the lymph channels. If morbid processes are going on in the tubes or lung tissue, irritating waste products have to be removed. If these can't pass freely along the peribronchial and interlobar lymphatics through the root channels, then they must tend to escape by way of the sub-pleural lymphatics into those of the Pleura, and so lead to a stagnation there. A continuation of this must tend to set up a chronic fibrotic process, however slight, in the lung tissue and pleura. In the Pleura it may

* (see. Virch. Arch. BD. CLXV Heft I). Watanabe's observations)

lead to a chronic Pleuritis and adherency. I am of opinion that this chronic interstitial fibrotic process is a much more frequent occurrence than is generally supposed, and that it may occur in some cases of prolonged chronic Bronchitis, apart from any marked primary lung disease I firmly believe (Pleuro-Bronchitis). (See Drummond, quoted by Ewart, Allbutt's System of Medicine, page (64)).

I am inclined to look upon this process as the method of production of the initial Pleural adherency, especially in those cases which I have described in the section on Morbid Anatomy as chronic Bronchitic Bronchiectasis, and it may be the mode of production in other cases as well.

Associated lung conditions in Bronchiectasis:

I find in my cases that dilatation of the tubes may exist in lung tissue which may present varying degrees of fibrosis or consolidation and also in normal lung tissue. Some authorities as Corrigan, Rokitansky and Lebert hold the view that the tubes are in part dilated by the contraction of the surrounding fibrotic lung tissue, and in part by inspiration. Accepting this view, as the mode of production in some cases, it cannot explain those which occur in fairly normal lung tissue. Their view as to the part played by inspiration in dilatating the tubes during fibrotic contraction seems to me untenable. In most of the cases I have seen, where clinically one has diagnosed a fibrotic condition of the lung, even in early cases, the inspiratory effort was much diminished and occasionally almost nil. I have carefully studied these cases as to the degrees of chest expansion during inspiration, and also watched the play of the diaphragm on the affected side under the X Rays and in most cases the former and the latter were diminished, in some cases markedly so. That forced expiratory effort with closed glottis might help in the dilatation of these tubes, which are said to be pulled out by the surrounding contracting lung tissue I can understand, but that diminished inspiratory effort which I hold is usually associated with such conditions can have this effect I very much doubt. All the tendencies in such a condition are towards a contraction of the tubes,

such as happens in Tubercular cavities undergoing contraction as a result of a healing process. It is logical to think that if the surrounding lung tissue is contracting through a fibrotic process, the tubes are in the same condition also and the fact that Inspiratory effort is diminished leads one to suppose that this process in the tubes would be in a still more favourable condition for contraction. That increased expiratory effort $\overline{C}$ closed glottis, however, might counterbalance this contraction of the tubes, I can well believe. Biermer's contention that Pleural adhesions increase the action of the Inspiratory force and so cause dilatation, is utterly opposed to all my clinical observations on this point.

A point of great importance in this discussion is, as to whether this fibrotic condition is primary or secondary. This is a very difficult matter to determine. By the exponents of Corrigan's theory it is held that it is a primary condition starting in the pleura and extending to the Pulmonary interstitial connective tissue, and so leading to its contraction and subsequent dilatation of the tubes. There are no true grounds for such an assumption, and I believe that in most cases the fibrosis in the lung is coincident with or apart from the associated pleural condition. I have seen a case post-mortem which presented slight dilatation of the tubes with pleural adherency and commencing fibrosis of the lung tissue, contracting and pulling asunder the layers of the pleura attached to the chest wall, with no microscopic appearances of any invasion of the lung tissue by a fibrotic extension along the interlobar and intralobular septa. I am strongly of opinion that in the great majority of these cases the fibrosis is primary in the lung, apart from the pleural condition. That fibrosis may be occurring in the Pleura at the same time I don't doubt, but it may exist apart from any such pleural condition whatsoever. I admit that fibrosis may and does take a part in the subsequent further dilatation of these tubes, but the initial dilatation existed before the fibrotic lung condition.

My view is then, that fibrosis of the lung takes no part in producing the initial dilatation of tubes, whatever results it may have later (See Drummond, quoted by Ewart, Allbutt's System of Medicine, page (64)). Kingston-Fowler who is opposed to this theory of fibrosis of the lung causing the dilatation,

gives an example where the wall of a Tubercular cavity has been made irregular at one point, by,-it might be inferred,- a fibrotic septum passing in from the pleura and so drawing on the walls of the cavity at that point; the real explanation in all probability being that this irregularity was produced by the cavity itself undergoing contraction and so pulling on this septum. I was able to obtain at a post-mortem in a case of Saccular Bronchiectasis, a specimen of an exactly similar condition and as it is of some interest I add a microphotograph of it. (See microphotograph No. (XXVII)). The case in which it was found was that of A. S. Fenner, whose physical signs, appearances of the chest under the X Rays and temperature chart I add at the end of my paper. Throughout the right lung were a number of terminal saccular Bronchiectases, globular in shape, varying from the size of a small walnut up to a hen's egg. In one saccular cavity a thickened fibrous septum was seen passing in from the thickened pleura, which had been adherent to the parietes, to its wall. In comparing this condition with the other globular cavities it was obvious that the cavity was rendered irregular in shape by its own contraction,and not by the septum. I add a microphotograph shewing this interesting condition (See microphotograph No.(XXVII))

The results of my studies in this most difficult problem, lead me to the following conclusions.

(1) That Pleural adherency, occurring either locally or generally is a factor of marked importance in the initial production of Bronchiectasis, however slight, chiefly through the support which it thus gives to the lung tissue during continuous expiratory effort with closed glottis, especially when associated with diminished Inspiratory effort, as is often the case in such a condition. It may be stated that such slight adherency could not alter the diffusion of air-pressure to any marked degree, but I am bound to say that all my enquiries lead me to believe that it does so.

In some cases under almost exactly similar conditions, we may get the production of Emphysema only and in others Bronchiectasis. The only factor which I can find which seems to determine in most cases, whether the one or the other shall occur, is that of Pleural adherency.

I don't believe that Pleural adhesions are the initial cause of bronchial dilatation, by traction on the tubes, directly or indirectly, because as

already mentioned, Kingston Fowler points out that at least in some cases, pleural adhesions do not exist, also I have examined specimens of Bronchiectatic lungs where, although the pleura was adherent, no fibrotic process extended from the pleura into the lung tissue. That the fibrosis of the lung tissue may play a part as a secondary process in maintaining or even increasing to some extent the existing dilatation I firmly believe, but I am of opinion that it is coincident with, or secondary, to any associated chronic pleuritis.

My enquiries also lead me to accept the view, that pleural adherency having occurred, commencing interstitial fibrosis in the lung tissue causing contraction, pulls apart the layers of the pleura, which subsequently becomes thickened by the interpolation of a gelatinous material which ultimately becomes fibroid. I have seen this occurring in a case, where in the least affected lung, with pleural adherency, commencing fibrosis of lung tissue was causing separation of the pleural layers. (See Microphotographs Nos. (21 & 22)).

(2) Pleural adherency alone, in cases where the disease is more advanced, specially where the pleura is thickened, leads to a greater diffusion of the air pressure away from the terminal parts of the respiratory tracts and so tends to a greater degree of dilatation of the larger tubes, i.e., cylindrical Bronchiectasis. I have noticed that in some cases where the variety of Bronchiectasis was saccular with cylindrical, there was marked adherency with marked thickening of the Pleura, and in other cases with purely saccular dilatations, that the pleura was only slightly adherent or non-adherent (Pleuritic types).

In all my post-mortem cases of Pure Bronchiectasis, I find only one in which Pleural adherency was absent over the dilatations, and although it is a difficult matter to determine whether or not adhesions were present from the onset of the disease, nevertheless its occurrence is highly significant.

I incline to the view that in some cases, Bronchiectasis seems to be produced without the intervention of Pleural adherency, but I am strongly of opinion that Pleural adherency undoubtedly is an important factor in the initial dilatation of the Bronchial tubes.

Treatment:-

Through the kindness of the Physicians at the Brompton Hospital, I have been able to make an enquiry as to the comparative value of various remedies in the treatment of Bronchiectasis; and combined with a study of the records of treatment here, I have obtained a fair estimate of these. The methods I wish to describe are the following:-

1. Creasote Vapour Baths.
2. Subcutaneous injections of Guaiacol.
3. Intra-tracheal injections of Guaiacol.
4. Intra-Venous injections of Formalin.

The points I wish to refer to specially, are the effects of these various remedies on the general condition of the patient, cough, foetor and the amount of expectoration, temperature, weight, and the mental condition. Apart from the statements made below, a short note of each case along with the temperature chart, shewing the effects of the various remedies will be found at the end of this paper.

Creasote Vapour Baths.

It is unnecessary to give here a full detailed account as to the method of giving these baths at this hospital, as this will be found fully described in Fowler and Godlee's book on Diseases of the Lungs, page 138. The duration of the baths varied from 20 minutes to 1 hour.

Effects on the cough: and amount of sputum:-

The treatment of my cases by this method shews that while the cough itself is lessened, the patient enjoys a longer interval of freedom from it. When the baths are first given, the amount of expectoration is usually increased, and thereafter in favourable cases there is a steady and constant diminution in the amount (see Case C.C. No. I *~~and W.R. No~~.). If the baths which have been continued for some time, are taken off, there is usually an immediate diminution in the amount with a subsequent rise. However in unfavourable cases there is no effect produced at all, or on the contrary there may be an increase.

*: Refers to No 9. Daily Temperature Chart

Effects on the Foetor of the sputum:-

In most cases the foetor is much lessened and in some it entirely goes (as in Cases C.C. No (I)* and H. S. No. (IV)*). However in very unfavourable cases it remains as before.

Effects on the temperature:-

In the great majority of cases the temperature undergoes an immediate lowering, and usually continues so. It also becomes more regular and less remittent. If the baths which have been continued for some time are stopped, the temperature may become irregular and remittent (see Case W. S. No. (15)*) - this is an extremely bad sign in the treatment of these cases by this method. One fallacy comes in here and it is this, that often when the patient is admitted he has a slightly elevated temperature which may continue for some time, but if he is allowed to rest, without any active treatment, the temperature in some cases returns almost to normal. This is well shown in the case of D. Farrendon No. (X).* This is a point which is overlooked in describing the effect of such treatment on the temperature. Generally it may be stated that there is a uniform ratio between the effect on the sputum and temperature, but this is by no means constant; the temperature sometimes being lowered and at the same time the amount of the expectoration increased.

The occurrence of inverse types of temperature is also to be noted here. This type of temperature is usually looked upon in acute miliary tuberculosis with grave suspicion as heralding a fatal issue, but here it occurs in some of the most favourable cases.

General condition of the patient:-

In most cases this is considerably improved, but this is by no means constant.

Mental condition:-

This is an effect which is generally overlooked. The Bronchiectasis case is usually of the melancholic

* refers to No. of daily Temperature Chart at end of paper.

type, but I must say that the effects of these baths in many cases is to render him much more so.

Weight:-

In nearly all the cases the weight is increased, in some very markedly so. (See weights of cases reported in the charts).

It is of interest to note the effects of two or more baths.

Case H. D. No. (VIII) * shews a great increase in the amount of sputum, as the result of a daily bath, but when 3, 4, or 5 daily baths are given the amount is much diminished. Note also the great diminution in the amount of sputum when the baths are taken off. Curiously enough Case J. C. No. (VI) * shews almost the opposite effect, i.e., a slight increase with one bath daily and a great increase in the amount of the sputum when two baths daily are given. The diminution in the amount when the baths are discontinued is noteworthy as also its subsequent rise when the two baths daily are continued.

Apart from its disinfectant and bactericidal qualities creasote is an admirable stimulant expectorant in these cases, but when pushed too far this last quality seems to be much lessened and consequently part of its value as such, lost. However I should be inclined in every case during a short period of its treatment to increase the frequency of the baths to 2-4 times daily as the case may be, and so bring the bactericidal properties chiefly to bear on the septic dilatations. I am strongly of opinion that this would lead to better results.

The case of Caroline Cooper No. (I) * whose temperature chart with a few short notes of her case are given at the end of this paper, is an example of a complete permanent arrest. This patient presented herself to me for re-examination two weeks ago and I then had an opportunity of noting the physical signs and of taking a screen and X Ray photograph of her chest, which I add to my paper (See X Ray Photo No. (246). ✠ She was then in good health with no cough and no expectoration, and is attending school.

It is worthy of note that the cases which give the most unfavourable results are those of tubercular Bronchiectasis. (See cases W. Spearman No. (15) and J.C. A.. No. (14.B)).

✠ also see X' Ray Notes: No X

* refers to no. 8 daily temperature chart/

Subcutaneous injections of Guaiacol:-

In favourable cases the effect on the cough is usually very decided, causing it to be easier and not so constant. The amount of the sputum is increased at first with a subsequent constant and steady diminution. The temperature is lowered, but the irregularity may continue. The effect of adding creasote baths to the injections are well shown in cases J. H. No. (III)* and A. W. No. (VII)*, by causing the temperature to be lowered and become more regular with a marked approximation of the morning to the evening temperature. In the case of J. H. No. (III)* the doses of injection of sterilized oil of Guaiacol in almond oil (1 in 4) was m II daily increased to 3.C.C's. The sites of injection was into the two buttocks alternately. I may mention that the injection caused no local trouble whatever - no obvious inflammation and no abscess formation. In severe types of Bronchiectasis it has been found that these small doses sometimes gave unsatisfactory results. A case of this type (Case A. W. No. (VII)*) came into hospital here in September of last year under the care of Dr. Kingston Fowler. It was diagnosed as chronic Pneumonia (?) with probable Bronchiectatic cavitation. The sputum was intensely foetid, and no tubercle bacilli were found in it on repeated examination. It was decided to try the effect of large doses on this case. The initial dose was m XXX of sterilized oil of Guaiacol (in almond oil) 1 in 5, daily, and it was increased up to m 150. The injections were almost exclusively made into one buttock. The patient was too ill to be out of bed and in a very melancholic condition. The effect on the sputum was a slight and steady decrease in its amount, with a marked lessening of the foetor. At times the foetor would be absent for one to two weeks or more, and then would return for a day. At first the temperature was somewhat lowered and became more regular, but as the doses increased it became more irregular and of a more remittent type. Later on when creasote baths were added to the injections, the temperature fell almost to normal and the amount of sputum was increased. The most marked effect of all was the great improvement on the general condition of the patient, especially the mental condition. I have never seen so marked an improvement in any such cases however mild. The improvement continued and she was able soon to be taken down to take the creasote baths. I have to report however that the patient suddenly expired, while

*: refers to No. of Daily Temperature Chart.

sitting up in bed taking her tea. On post-mortem examination no cause could be assigned for the sudden death, but the condition revealed was that of chronic pneumonia (unilateral) with a large gangrenous bronchiectatic cavity filled with foul smelling, pultaceous material.

The improvement was so marked in this inveterate case that I should feel justified in again trying these larger doses, and in this opinion I am supported by Dr. Kingston Fowler.

One interesting point of practical importance in connection with this case is worthy of mention, and this is the local effect of the injections into the buttock. Formerly when this method of injecting large doses of Guaiacol had been tried, it was discontinued in many cases because of the invariable abscess formation which occurred at the seat of injection, though the buttock was slightly tender and shewed some degree of hardness to the touch, no abscess formation occurred, and the number of injections amounted to 52, This was a point of some practical interest as I had an opportunity at the post-mortem examination of cutting into the buttock and observing the state of the tissues where the injections were made. I found these tissues tough and hard, evidently in a state of subacute inflammation with some small haemorrhages, but there was no evidence of any pus formation. I add a microphotograph of this buttock tissue. (see microphotograph No. (XX)α/β.). It should be mentioned that the buttock was thoroughly disinfected with carbolic acid poultices for some days prior to injection and kept scrupulously clean. The hypodermic needle was boiled every time before use, and the Guaiacol solution was specially prepared in small quantities every week. I took the opportunity of making two series of plate cultures from this Guaiacol solution and every plate remained sterile.

Therefore with strict antiseptic precautions, in most cases one should never have to discontinue the treatment on account of abscess formation at the seat of injection, unless in exceptional cases.

Intra-tracheal Injections:

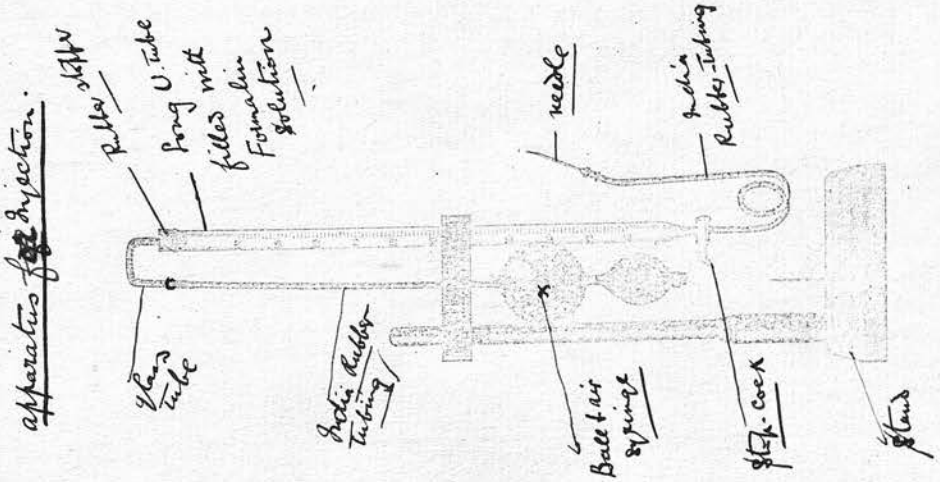
This method so strongly advocated by the late Sir Thomas Grainger Stewart, Colin Campbell and others, is of much value in some cases. It has almost been given up here, but I think there is no doubt as to its value in some cases and is well worthy of a trial combined with Creasote Vapour Baths.

I record the case of G.S. No. (II).*, giving the results of this combined method of treatment, with one injection and one Creasote Bath daily and also two injections and two Creasote Baths daily. The effect of the daily Intra-tracheal injection alone was to slightly diminish the amount of the sputum, the temperature remaining much about the same. The foetor of the sputum was considerably lessened, thereafter the addition of a daily creasote bath to the injection did not produce much change. Two injections and two creasote baths daily produced an increase in the amount of the sputum generally, with no decided effect on the temperature. The most marked effect was on the extreme foetor of the sputum which entirely disappeared and the general condition was improved. The dose of injection was, ~~3~~ of menthol, 10 parts Guaiacol 2 parts, ol-olivae 88 parts. 3ss/.

Intra-Venous Injections of Formalin:

This method of treatment was introduced into the hospital here by Dr. Maguire for the treatment of Pulmonary tuberculosis. It is too early yet to give any judicious view whatever as to its success or non-success in that disease. But the method appearing to be founded on a rational and scientific basis it was resolved to give it a trial in a case of Bronchiectasis.

The apparatus for injection consists of a stand, holding a long glass tube with a stop-cock at its



lower end. The upper end of the tube is fitted with an india-rubber stopper which is perforated by a glass tube to which is attached a ball and air syringe.

* refers to No. of Daily Temperature Chart/

To the lower end of the long glass tube is attached a piece of india-rubber tubing about 8 inches long, and fitting on to the end of this is a long needle.

The stop-cock being turned off, the long tube is filled with the Formalin solution which just prior to use has been slightly warmed. The stopper is then put into the upper end of the long tube and the apparatus is then ready for use.

The patient's arm is then thoroughly prepared, as if for operation, over the regions of the median cephalic and median basilic veins. A lightly applied tournique (handkerchief) is put around the upper part of the upper arm, and the patient is made to grasp firmly a roller bandage to render the superficial veins more prominent. The stop-cock is first of all turned on to allow the Formalin solution to completely fill the needle and rubber tubing attached to it. The needle is then put into either the median cephalic or median basilic vein, the tournique being then removed. With the aid of the ball-syringe the Formalin solution is gradually forced into the vein, and after the required amount has been run off, the needle is withdrawn and a small pad of lint held over the seat of injection for a couple of minutes or less, and thereafter the patient may use his arm as he pleases with no bad effects. The time of injection varies, but it should never occupy more than 1 to 2 minutes. With a larger needle (which is really less painful) the Formalin solution (50.CC), can be injected in about eleven seconds. I regard this latter of decided advantage and value in increasing the effect of the solution.

The Formalin solution should never be strongly heated as it decomposes. The formula consists of a 1 in 2000 solution of Formic aldedyde in normal saline solution (.75%).

I should add that in giving the injection the strictest antiseptic precautions should be taken. The amount of injection is usually 50 CC). of a 1 in 2000 solution, given on alternate days during the first week, and daily thereafter. The dose may be increased to 65 CC with safety. I have seen and have personally injected many cases of phthisis by this method and I have never experienced any evil results whatever from its use. With some practice one can become quite expert in putting the needle into the vein, but at first it presents some difficulty.

The rationale of the method consists in injecting into the vein such a quantity of an antiseptic fluid which shall reach the lung, and so act on the micro-organisms causing the disease. Personally I have only treated one case of Bronchiectasis so far,

i.e., that of Frederick Lovejoy, a virulent case, who came into the hospital here under the care of Dr. Acland. The temperature chart with a few notes of his case I add at the end of this paper (see case F.L. No. (XII.))*. The dose of injection varied from 45 CCs to .60 CCs of a 1 in 2000 solution. The injections were combined with creasote vapour baths. There was at first an increase in the amount of sputum, but later, a decided diminution. The extremely foul odour of the sputum soon began to lessen and to disappear entirely for one to two weeks at a time, and at the end of treatment was generally very much less. The temperature was generally lowered and became more regular and not so remittent. The patient gained 10¾ lbs, during his stay in hospital (158 days) and his general condition was much improved, the patient himself being highly pleased with his progress. The physical signs on leaving were the same as on admission. One of the most marked effects, was on the taste - the previous foul taste disappearing for a long period and only returning towards the end of the course of treatment. These patients usually experience a foul taste in their mouths, which is a source of considerable discomfort, and relief from this was much appreciated by this patient. His stay in hospital however was suddenly cut short by having to be removed, owing to a maniacal fit to which he had been subject to for a short time previously (cerebral abscess-frontal lobe)*.

He was sent to an Infirmary and recovered in about a week. The patient being extremely anxious that the treatment should be continued, so convinced was he of the benefit that it had done him, was readmitted under Dr. Acland on March 5th 1902. His condition was much about the same as when he left hospital in December last. The physical signs shewed no alteration but the breath however was distinctly more foetid, and the amount of sputum increased. It was then resolved to give him the Formalin injections alone without the creasote baths to watch their effect. After I had injected him on ~~four~~ occasions his temperature began to rise and he was thought to be suffering from an attack of Broncho-pneumonia with probably some gangrenous spread. He became rapidly emaciated and his sputum became horribly foetid. Meanwhile I had stopped the injections. On the 35th day after admission he died.

The post-mortem examination shewed the case to be one of marked cylindrical Bronchiectasis in left lung with some slight Broncho-pneumonia in lower lobe of right lung. On examining the brain, an abscess about the size of a large walnut was found in the

* left hospital December 23rd-1901 / * refers to the No. of the daily temperature chart at the end

right frontal lobe. The temperature chart with a diagram of the chest signs and the notes of the chest condition as revealed by the X Rays will be found at the end of this paper.

The occasional occurrence of inverse types of temperature, both with creasote vapour baths alone and also combined with Formalin injections is to be noted in this case.

It is of course impossible to give any definite opinion, at present, as to the success or non-success of this method of treatment by Formalin Injections but it is well worthy of a further trial.

I add the temperature chart of an extremely virulent case of Bronchiectasis treated here by Dr.Maguire by this method. But as only 7 injections could be given, it is impossible to judge of its effects. (see Case J.W. No. (XIII) *).

As a result of my experience I would say, that the best results in the treatment of Bronchiectasis are to be had from the combination of creasote vapour baths with subcutaneous injections of Guaiacol in small doses (M VI to M X). Starting with this combination, I would give them once daily for the first few weeks, and during a period of at least a fortnight or three weeks I should increase the frequency of the creasote vapour Baths to 2 to 3 times daily as the case may be, one injection only being given during this period; thereafter reverting for the remainder of the course of treatment to the initial doses. In most cases, to be of any decided value the treatment must be continued for at least 3 to 6 months. In inveterate cases I should be inclined to give a trial to the larger doses of the Guaiacol (M XX to M120 according to its effects) in combination with the creasote vapour baths.

As for the Intra-Venous Formalin method, it is yet on its trial and I can therefore express no opinion at present but I trust that it will receive a fair trial, in the hope that it may be found of value in the treatment of this loathsome disease.

*: refers to daily temperature chart at the end of paper

The Value of the X Rays in Bronchiectasis.

It has been my endeavour to enquire into the X Rays in the diagnosis of Bronchiectasis. In the ordinary case of Bronchiectasis which is presented to us in hospital, there is usually not much difficulty in the diagnosis by the ordinary clinical methods. But there are some very early cases which present considerable difficulty and it has been my endeavour to find out if the X Rays would help us in clearing up these.

One may say that the surgical treatment of Bronchiectasis is almost a closed chapter, at present, in the history of medicine. But what has struck me in some of these cases which have been so treated and have come to the post-mortem table has been, the failure to reach the bronchiectatic cavity by exploration and by subsequent surgical procedures. It is also a matter of considerable difficulty to determine exactly as to the presence of such cavities in the lung. Therefore I considered that my time would not be wasted, in enquiring into these various points.

Before submitting the doubtful early cases of Bronchiectasis to the X Rays, I considered that it would be more judicious to find out what they revealed to us in well-marked cases and it is fortunate that I did so.

At the end of this section I have recorded my results. In 3 cases I was able to compare the appearances revealed by the X Rays, with those revealed on the post-mortem table. The diagnosis recorded in each case is that given by the Physician under whose charge the patient was. I have to record my indebtedness to Dr. Barry Blacker, who has charge of the X Ray department at the Brompton Hospital, for his kindness in allowing the cases to be X rayed, and also for his skilled opinion on the appearances presented.

I find that in Bronchiectasis as in all chest cases, the X Rays are of very little value. In fact I may say that as a general rule, what the stethoscope can't tell us, the X Rays fail to make any clearer. In my opinion, they are very much overrated, and are apt to lead one to erroneous conclusions in the diagnosis of chest diseases.

While I was House-Surgeon to the Royal Infirmary Edinburgh, I took occasion to note their value in surgical cases, especially in fractures and foreign bodies in various parts and I then considered that they were of decided value in these cases. During my medical Residency there, I also had the opportunity of observing under the X Rays several chest cases, in which the diagnosis was doubtful, i.e., cases where the diagnosis lay between Aortic Aneurism and mediastinal tumour, for example.

During my studies here at the Brompton Hospital, I have followed the courses of a large number of cases of chronic Pulmonary Tuberculosis and varied chest affections, especially those which have come to the post-mortem table, after they had been X'rayed. The cases in which one would wish assistance in diagnosis specially, are early cases of Pulmonary Tuberculosis and Bronchiectasis; in cases where there is doubt as to whether the condition is one of Aortic Aneurism or Mediastinal tumour or where the presence of a foreign body in the bronchus is suspected.

All my experience goes to doubt the value of the X'Rays in the diagnosis of suspected early cases of Pulmonary Tuberculosis, where there are no obvious physical signs. I have a case under my charge in hospital here at present, i.e. G. W. presenting no physical signs in the lungs, but the symptoms all point to the case being one of phthisis and gave a typical and marked tuberculin reaction, but the X Rays revealed to us nothing.

Another case H. B. under my charge here presented a difficulty as to whether it was one of mediastinal tumour or Aortic Aneurism. The X'Ray report was to the effect that it was one of Aneurism of the ascending Aortic Arch. The post-mortem examination proved the case to be one of mediastinal tumour (sareoma). I mention all the above facts, though they may seem to be apart from the subject under review, to shew how extremely careful one has to be in estimating the true value of these X'Rays, as against the "glowing" accounts and impressions one gets from the medical press.

In Bronchiectasis, I found my results extremely disappointing. To put it shortly, the X'Rays in all cases, with the single exception of foreign bodies in the bronchi revealed less to us than the stethoscope did. They invariably confirmed the presence of some morbid process, as shewn by an opacity, but as to its real nature - nothing.

As to revealing the presence of cavities in the lung, it utterly failed, owing I believe to these being completely hidden by the dense fibrotic condition of the lung tissue. I have only to point to the case of A. Sydney Fenner, recorded below, to confirm this statement. In that case cavities were present in the right lung as large as a hen's egg and were unrevealed by the X'Rays. I may mention an interesting point in the above case. The X'Rays shewed a curious diffuse mottling throughout the left lung, which I had never before witnessed in any of

these cases. On post-mortem examination this lung was found to be studded with diffuse racemose tubercles. There were no Tubercle Bacilli in the sputum on repeated examination, and no Tubercular condition in the lung was even suspected.

Nothing indicating the presence of dilated bronchial tubes was observed in any of my cases.

The X'Rays are of decided value in diagnosing the presence or absence of foreign bodies in the bronchi, especially in cases where the physical signs may be almost nil. But one has to be on one's guard here also, and in support of this I would point to the case of Charles Royds recorded below, where a foreign body was revealed by the X'Rays, but on post-mortem examination no such body existed.

I endeavoured to get these well-marked cases of Bronchiectasis X'rayed, when their bronchial tubes and cavities were full of retained secretion in the hope of shewing up the condition more clearly, but the results have proved negative.

In conclusion I would state, with an unbiassed mind, that at present anyway, the X'Rays in Bronchiectasis are of very little value to us, except in revealing the presence of foreign bodies in the bronchi.

Detailed Results:

As a result of my investigations I find, that in doubtful early cases of Bronchiectases, the X'Rays always confirmed, the presence or absence of a morbid process. The presence of a slight lesion - usually basic - was shown by a slight opacity in the normal chest shadow, and usually by an obscuring of the shadow of the diaphragm on the affected side. It is to be noted also that in many cases, the diaphragmatic excursion was diminished as compared with that on the sound side. Occasionally, in early cases where there was little doubt clinically as to the diagnosis, numerous "streaks" (like rays) were seen spreading out from the root of the affected lung, specially radiating in the lower lobe. This was well-marked in advanced cases. When this phenomenon was not seen, I am convinced that it was over-shadowed by the existing dense fibrotic lung tissue. I have suspected that these "radiating streaks", in all probability represented the bronchial tubes, but of this I am not yet certain. However, in this connection, one has to remember that occasionally this phenomenon is seen in cases of Pulmonary Tuberculosis.

The opacity, mentioned above, as confirming the presence of some morbid process in the lung, is well-marked in advanced cases, especially if the lung tissue is highly fibrotic. In these cases, as for instance in chronic Pneumonia, the opacity may be so dense as to obscure the ribs, and it is common in such cases to get the diaphragm entirely obscured, so that its action cannot be seen.

Another point of some interest is, that in some cases, near the root of the affected lung, one often sees small, dark, well-defined, almost circular shadows, about the size of a sixpenny piece, which one might look upon as foreign bodies. I have been unable to explain their occurrence, but I would state, that one such case which revealed these shadows, shewed calcareous glands at the root of the affected lung, post-mortem. This was the only case shewing this condition by the X'Rays, in which I had an opportunity of seeing the lungs post-mortem. That these dark shadows, which look like typical foreign bodies in the lung, may be due to the presence of calcareous glands, one can readily believe. Two cases which were X'Rayed and which came to the post-mortem table, i.e. A.Sydney Fenner & Frederick Lovejoy, (see X'Ray Notes, Nos.* (~~VII~~) ~~VIII~~), did not shew this condition, and post-mortem the bronchial root glands were indurated, and greatly enlarged, but not calcareous.

* see also X'Ray Photograph No. 249.

However, it is to be borne in mind that the opacity as shown by the X'Rays was extremely dense in these two cases.

The curious "diffuse mottling" appearance got in the left lung of A. S. Fenner, which showed on post-mortem examination, diffuse racemose tubercle is a point of considerable interest.

I could find nothing to indicate the presence of dilatation of the bronchial tubes, nor in most cases, saccular cavities, which undoubtedly existed.

(1) :-

Charles Royds : aet. 14 years : under the care of.

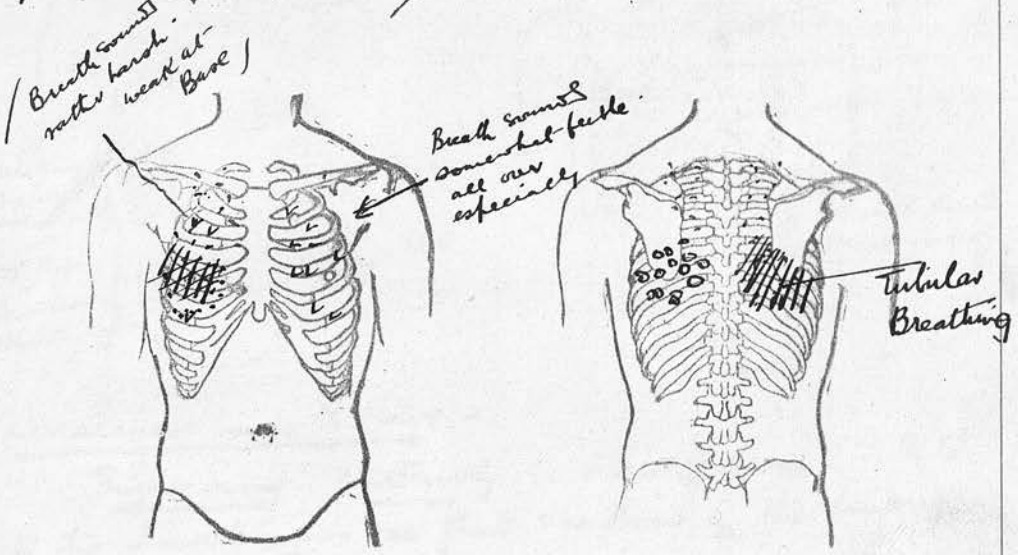
Dr. Hector Mackenzie.

Diagnosis : Bronchiectasis.

? Foreign body in the Bronchus.

No Tubercle Bacilli in Sputum.

Sputum very offensive.



appearances under the X'Rays :-

Mottled appearance of both lungs specially around and to the left side of the heart. In the middle of a small round shadow. on left side of apex of heart is a straight definite shadow. about 1½ inches long. ? Piece of Bone.

No cavity detected.

Post-mortem examination :-

No trace whatever of any foreign body. Marked cylindrical Bronchiectases both lungs specially of Right upper + lower lobes + also left lower lobes. Lung tissue generally slightly firmer than normal, otherwise healthy. Enlarged indurated glands at root of lung.

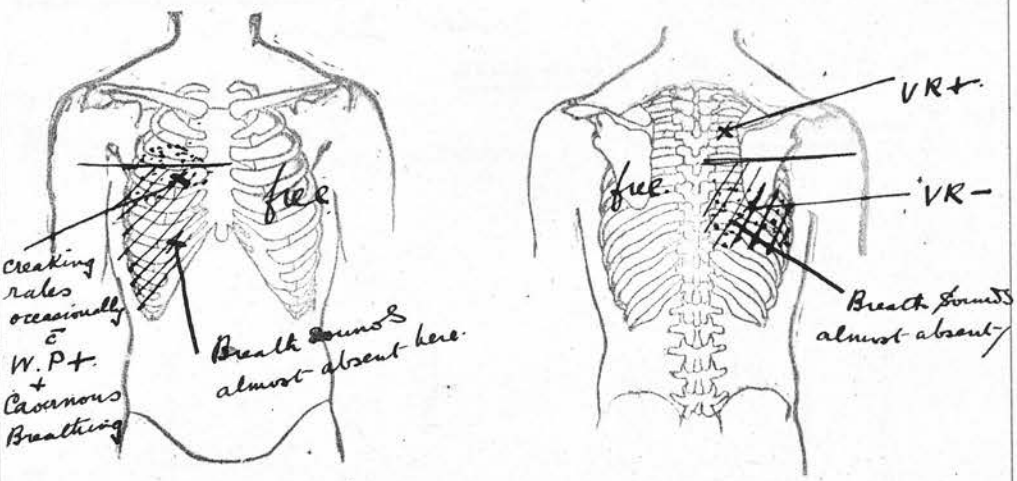
(see Lung Photograph XVIII).

(II):-

Samuel Egalton: aet. 29 years. under the care of Dr. Acland:

Diagnosis: Bronchiectasis? Right-Lung.

No Tubercle Bacilli in Sputum.
Sputum slightly Offensive.



appearances under X Rays:-

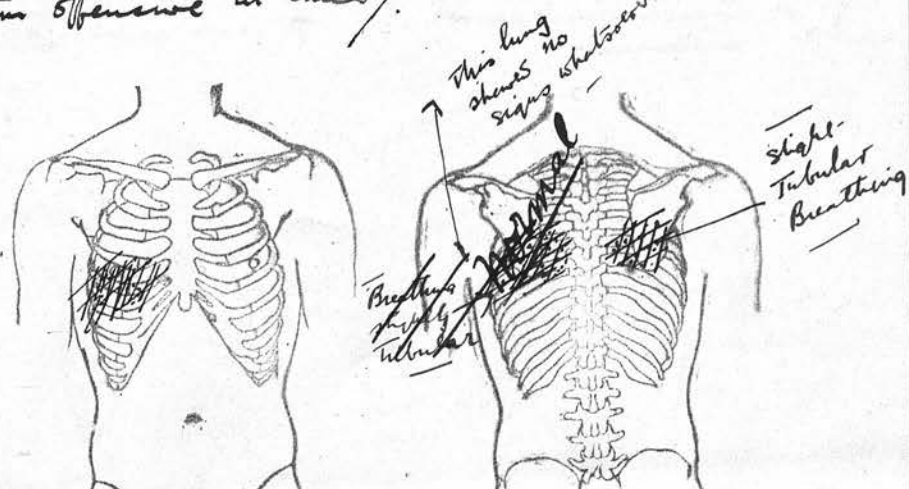
Right-Lung: anteriorly: opacity from 3rd Rib to the diaphragm so that shadow of the diaphragm was indistinguishable.
Left-diaphragm-movement-good.
Right-Lung: Posteriorly: opacity from 6th dorsal vertebra to base merging into shadow of diaphragm.
Heart: lies transversally.

(III):-

Frederick Hardwick:- aet. under Dr. Habershon.

Diagnosis: Bronchiectasis? Right-Lung.

No Tubercle Bacilli in Sputum
Sputum Offensive at times.



X' Ray appearances :-

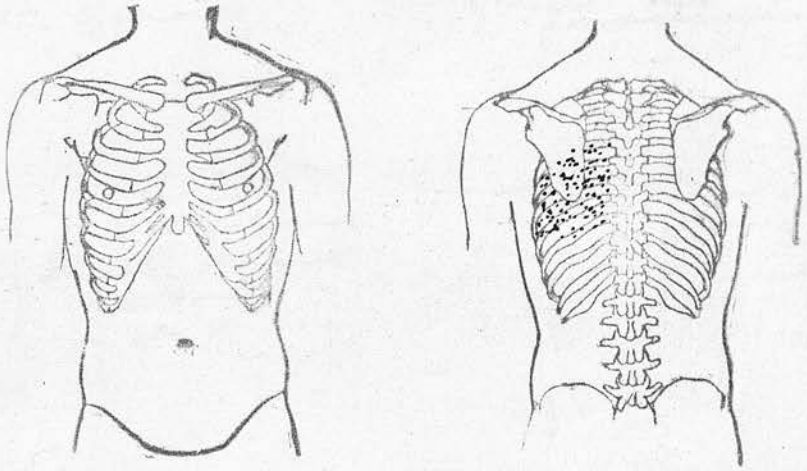
Patch in Right lower lobe (upper part).
separated from diaphragm by a bright-patch.
The former patch is irregularly opaque and about
4 inches by 1½ inches in size. /.

(IV) :-

Florence Harrison. aet. 30. yrs under the care of.
Dr Horton Smith.

Diagnosis: Bronchitis
Bronchiectasis ?

No. Tubercle Bacilli in
Sputum. offensive /.
clubbing of the fingers / marked /.



X' Ray appearances :

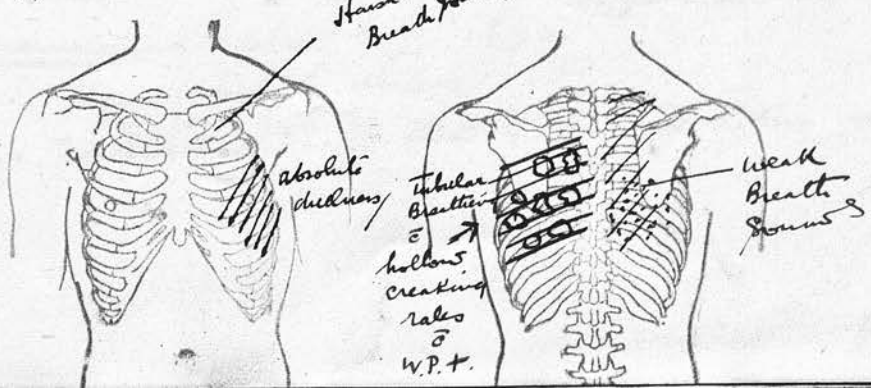
Middle of Right-Lung: Ribs obscured over
the region & are not so clear as usual. /.

(V) :-

Frederick Woodings: aet. 30 years. under the care
of. Dr Kingston Fowler. /.

Diagnosis / chronic Pulm^y Tuberculosis.
? unresolved Pneumonia.
? Bronchiectasis.

Tubercle Bacilli in
Sputum
Sputum offensive.
Had Pneumonia (lobar) (left) Left upper & lower lobes.
6 months before meet. ? Pleural adhesions on right
illness / side. /.



X' Ray appearances :-

Left Lung : nearly opaque. less so at apex.

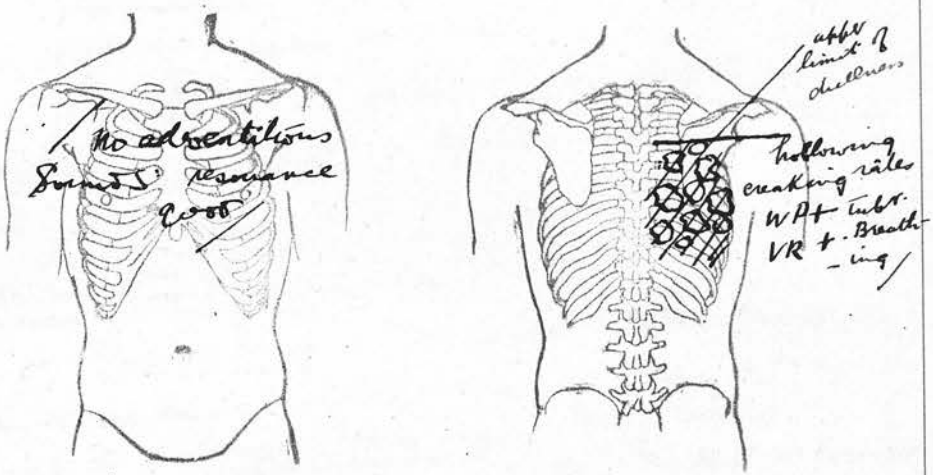
Right Lung : apex is very brilliant. ? cavity nothing suggesting Basal adhesions diaphragm moves freely /.

Seen

(VI) :-

Morris Hellover ; aet. 21 years. under the care of. Dr. Habershon. + late Dr. Kingston Fowler.

No Tubercle Bacilli in Sputum Sputum Offensive / diagnosis / ? accumulated Bronchiectasis of Right lower lobe.



X' Ray appearances :-

Right Base : dark shadow from 8th dorsal Vertebra to Base.. shadow obscuring the ribs. no fluid movement. diaphragm obscured. whole of lung is somewhat opaque. Pneumonic ?.

Left Lung : did not appear to be quite normal.

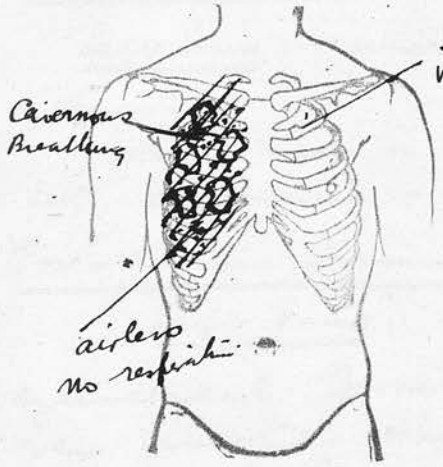
(VII) :-

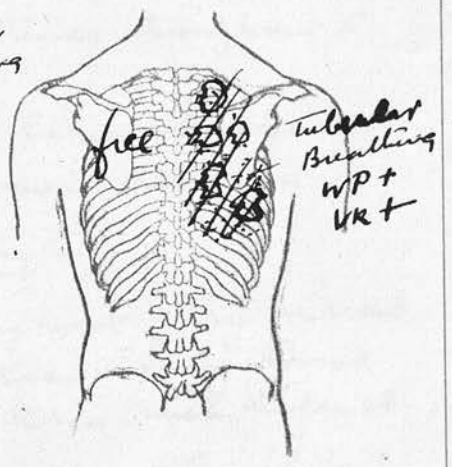
A. Sydney Tenner ; aet. 16 years under the care of. Dr. Percy Kidd.

No Tubercle Bacilli on repeated examination. diagnosis : Right Lung / chronic Pneumonia / Sputum extremely offensive / Bronchiectasis /

Pneumonia (lobar) Right) : 1 year before onset of illness /

(See temperature chart added to my paper. of this case).





X'Ray appearances:

Right Lung: opaque throughout. ribs seen. diaphragm not seen.

Left Lung: full of a distinct diffuse mottling. diaphragm not clearly seen.

Post-mortem examination:

Right Lung: Throughout whole lung. large cacular cavities varying from a large walnut to a large hens egg. lung tissue extremely fibrotic throughout. marked adherence of Pleura.

Left Lung: Throughout whole lung was a diffuse racemose tubercular affection. No Bronchiectasis. Pleura adherent generally.

(VIII) (George) Lovejoy: aet. 21 years, under care of Dr. Acland.

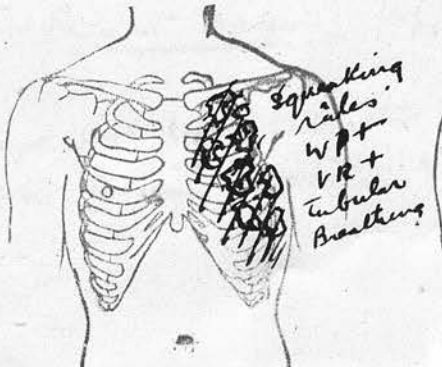
Frederick

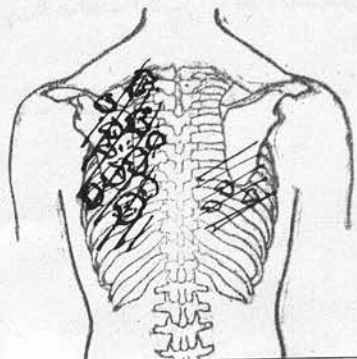
No. Tubercle Bacilli in Sputum.

Sputum offensive.

Diagnosis: (see temperature chart added)

Bronchiectasis: Left Lung. ? Right lung





X Ray appearances:

Left Lung: numerous streaks throughout & opacity.
Dark mass in both larger bronchi.
Left diaphragm: - movement deficient.

Post-mortem examination:

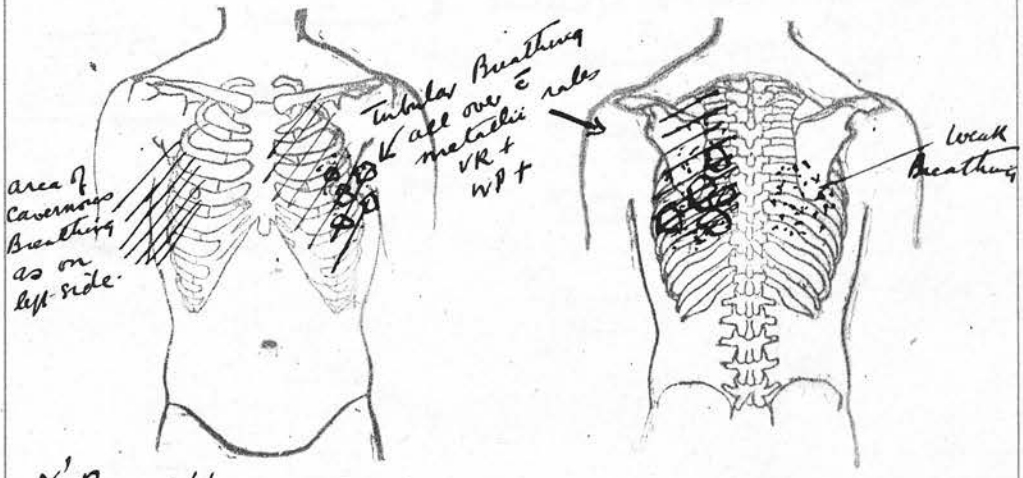
Left Lung: throughout very marked cylindrical Bronchiectasis. Lung tissue very fibrotic. Dilated tubes much thickened.

Right Lung: Some slight Broncho-Pneumonia in lower lobe: otherwise spongy & edematous.

(IX):-

Alfred Eldridge: aet. 3½ years under the care of Dr Adams. Diagnosis:-

No Tubercle Bacilli in sputum. Bronchiectasis: sputum very offensive.

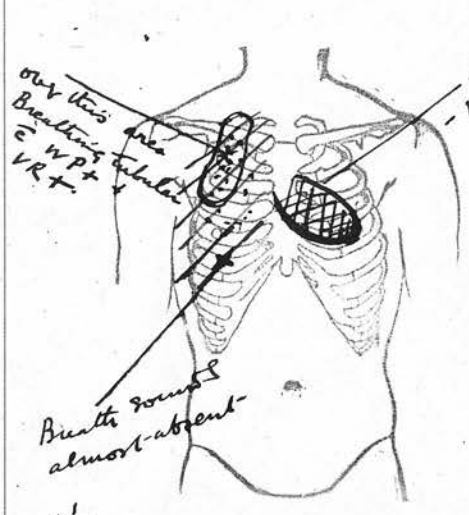


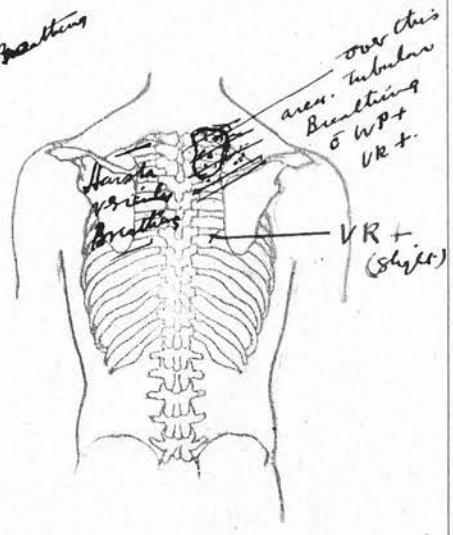
X' Ray appearances:-

Left lower ⅔ Lung: filled & branching opacities.
Right Base: Same as above.
Right apex: apparently normal.

(I have just heard that this patient dies soon after leaving hospital.)

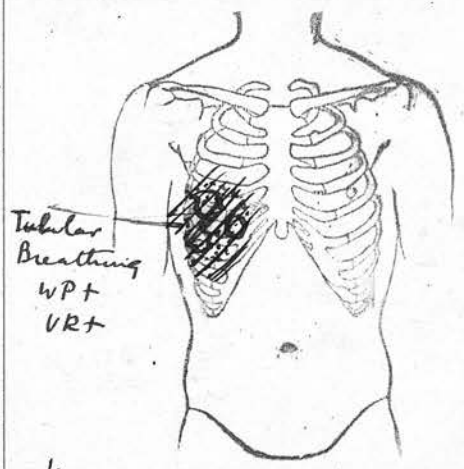
(X) :-
Caroline Cooper : aet: 12 years : under the Care
of Dr. Percy Kidd.
No. Tubercle Bacilli in Sputum : Diagnosis :
Sputum Offensive / Bronchiectasis.

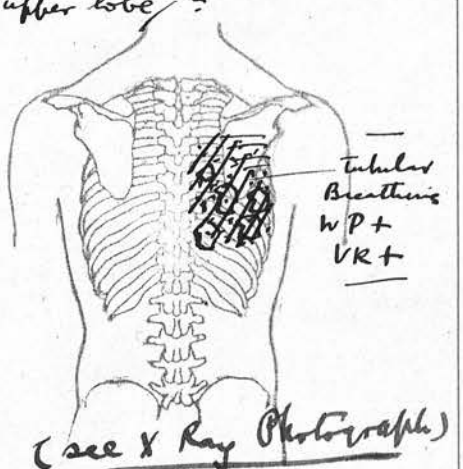




X Ray appearances :
middle part of Right Lung obscure.
(See temperature chart added.)
This is the case quoted of Complete Permanent-
asset- by treatment- & Creasote Vapour Baths.
(See X Ray Photographs)

(XI) :-
Daisy Farrandon : aet. 14 years : under the
Care of Dr. Green.
No. Tubercle Bacilli in Sputum examined carefully on 12 separate occasions
Sputum Offensive / Diagnosis : Bronchiectasis.
{ Quiescent tuberculosis left upper lobe ?





(see X Ray Photographs)

X Ray appearances :
upper part of Right Lung more opaque
than left.
Striae on both sides - external to heart.
No cavity : No Consolidation seen.
Both diaphragms - first fair / movement

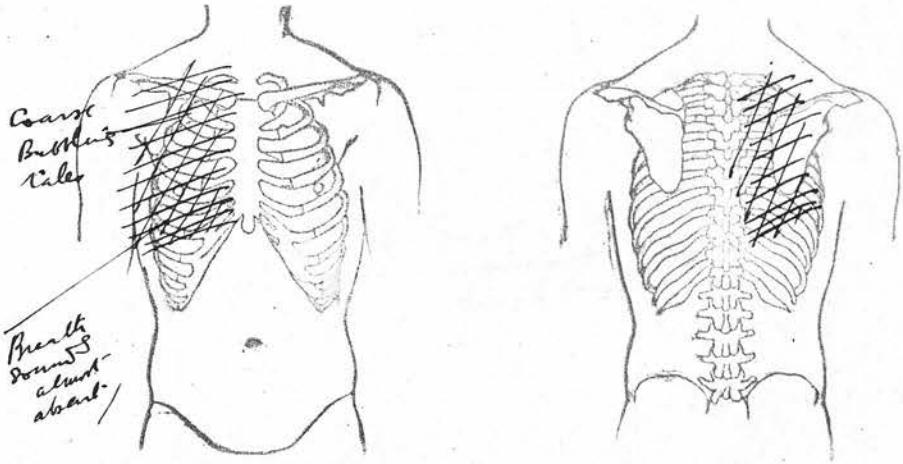
XII :-

Charles Sumbrey: aet: 23 yrs under the care of Dr. Percy Kidd. diagnosis...

No Tubercle Bacilli in Sputum

Sputum Offensive

Bronchiectasis.



X Ray appearances:

Right. lung: some opacity general. very slight movement of diaphragm

Left lung: normal. diaphragm moves well.

XIII :-

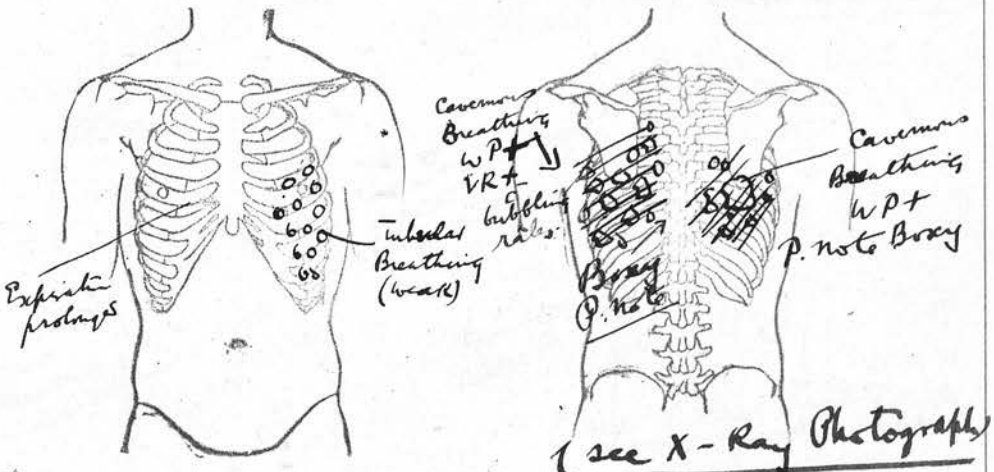
Mabel Hewitt. aet: under the care of Dr. Percy Kidd.

diagnosis:

No Tubercle Bacilli in Sputum

Sputum Offensive

Bronchiectasis left. lung



(see X-Ray Photographs)

X Ray - appearances:

Left lung: Generally obscure. diaphragm not clearly seen.

Both lungs full of streaks.

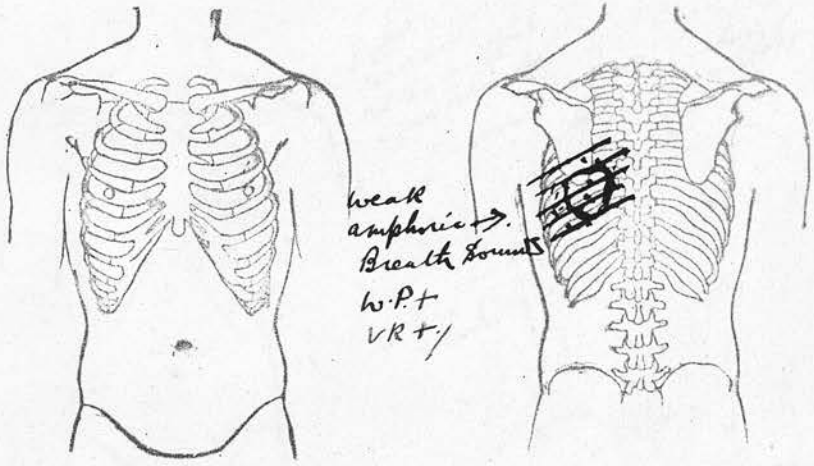
XIV :-

ada Whitehead : aet. 16 years : under the care of Dr. Percy Kidd.

No. Tubercle Bacilli in Sputum.

Sputum offensive.

Diagnosis : Bronchiectasis left. lung



X'Ray appearances :

Left. apex less opaque than the right- from spine of scapula to base, on left-side ribs are only just- discernible.

XV :-

alice Norton : aet. 8 1/3 years : under the care of Dr. Habershon.

No. Tubercle Bacilli in Sputum

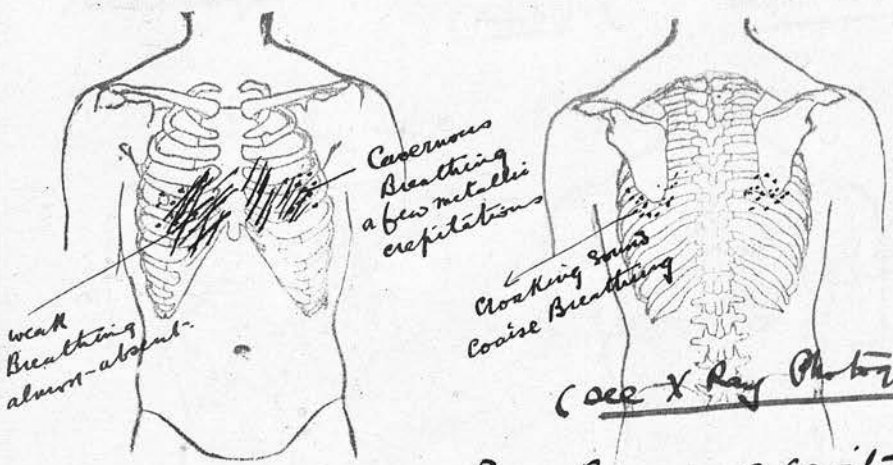
Sputum offensive

Diagnosis :-

Right. middle lobe.

Left- Base. Pleurisy

-: Bronchiectasis



(see X Ray Photographs)

X Ray appearances :- Left. Base : a cavity.

Right. Lung : some shadows in upper part. / Right- diaphragm deficient- movements
Left

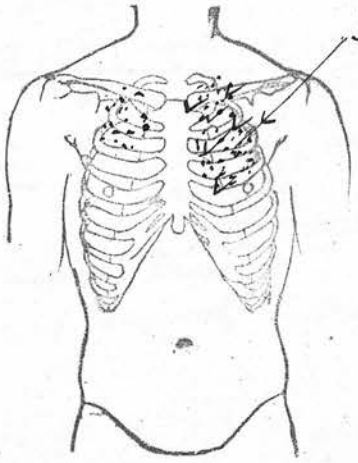
XVI :-

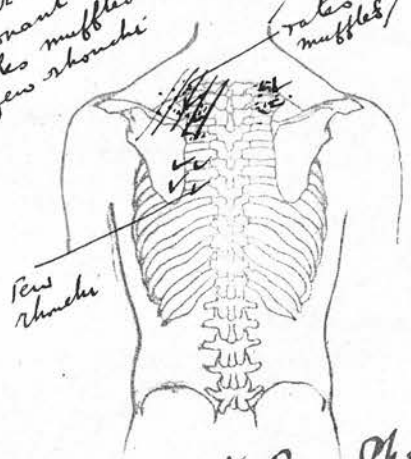
John Evans : aet. 25 years. under the care of Dr. Percy Kidd.

No Tubercle Bacilli in Sputum examined on 9 separate occasions carefully

Sputum Offensive

Diagnosis :- Chronic Bronchitis emphysema Bronchiectasis





(see X Ray Photographs)

X'Ray appearances :-

Both apices obscure.

Right-lung : Considerable General clearly throughout.

XVII : Alfred Smith : aet. 36. under the care of Dr. Acland.

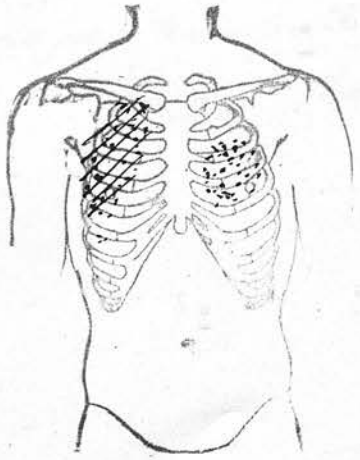
Diagnosis :

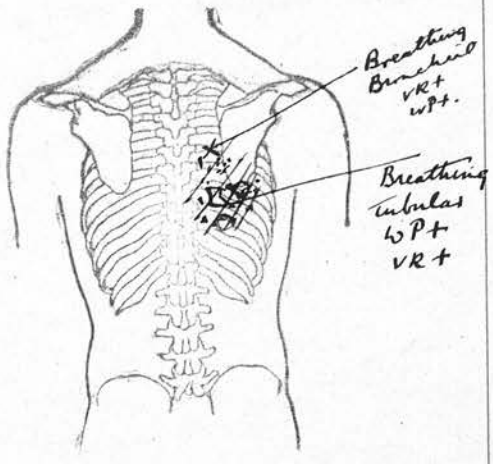
Tubercle Bacilli in the Sputum

Sputum not offensive.

chronic Pulmonary Tuberculosis

Illness began ō Pneumonia (lobar) (double) : ? Bronchiectasis Right-lower lobe





X Ray appearances :-

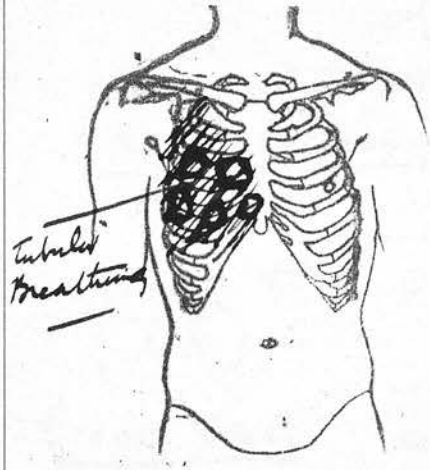
Left Lung : Root opaque.
Left diaphragm - movement slightly diminished /
Right Lung : Posteriorly : Base opaque
diaphragm fixed.

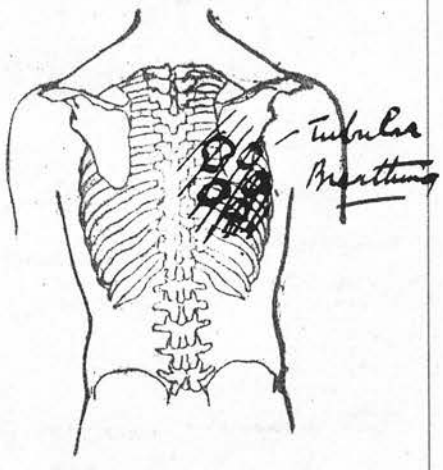
XVIII :-

Lucy Squires : aet. 4/12 years / at school /
under care of Dr Habershon

Sputum : large quantities very offensive /
No. Tubercle Bacilli in sputum
Fingers slightly clubbed /

Diagnosis :-
Bronchiectasis
(chronic Pneumonia)





X Ray - appearances :

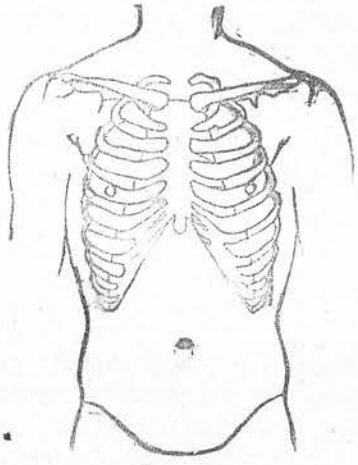
Right Lung :-
all over the shadow opaque, specially so over lower lobe /
Heart : Considerably displaced to right-side. /

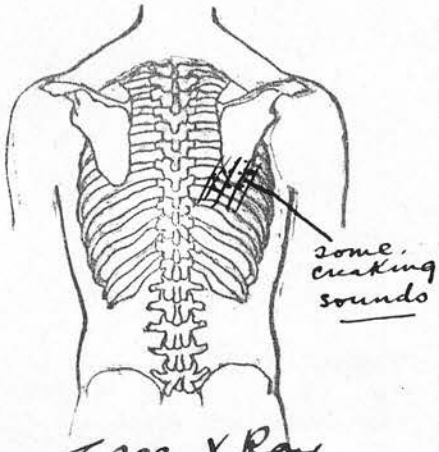
XIX :-

Joseph Burlington : aet. 5 8/12 years
under the care of Dr. Habershon

diagnosis : ? Early Bronchiectasis
Right lower lobe

/ sputum very offensive
no Tubercle Bacilli in sputum /.





(see X Ray Photograph)

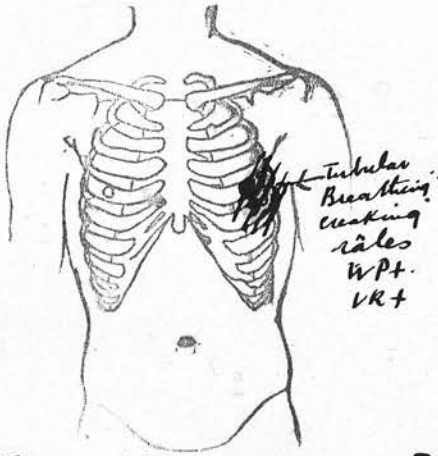
X Ray appearances :-

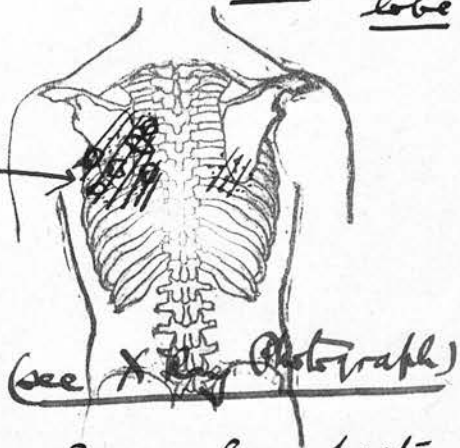
Right Base : slight opacity there : diaphragm not moving well.

XX :-

Grace Deely : aet. 6 9/12 years under the care of Dr. Habershon.

diagnosis : Bronchiectasis ? Right lower lobe
Left lower lobe





(see X Ray Photograph)

X Ray appearances : Left Lung : lower part distinctly opaque. diaphragm obscured.
Right Lung : / nothing abnormal

The following photographs illustrating my paper, which have afforded me much valuable help in my enquiries, were taken at the Royal College of Surgeons, and comprise all the more important specimens contained in the Museums of the Brompton Hospital for Diseases of the Chest, St. Thomas's Hospital, and the Royal College of Surgeons. One specimen from the Museum of the Victoria Park Hospital for Diseases of the Chest is added.

I have to record my indebtedness to the Physicians of these Hospitals and to Professor Stewart of the Royal College of Surgeons, for permission to study these various specimens.

The specimens were immersed in a water-tank and photographed there.

(I)

C

Specimen :-

Lung : shewing a typical, marked condition of Cylindrical Bronchiectasis. The circumference of the dilated tubes varies from ½ an inch to 1½ inches.

Note : marked prominence of circular muscular fibres in the dilated tubes especially near the root of the lung.

Lung tissue : extremely fibrotic.

(St. Thomas's Hospital Museum)

(II)

d

Specimen :-

Lung :

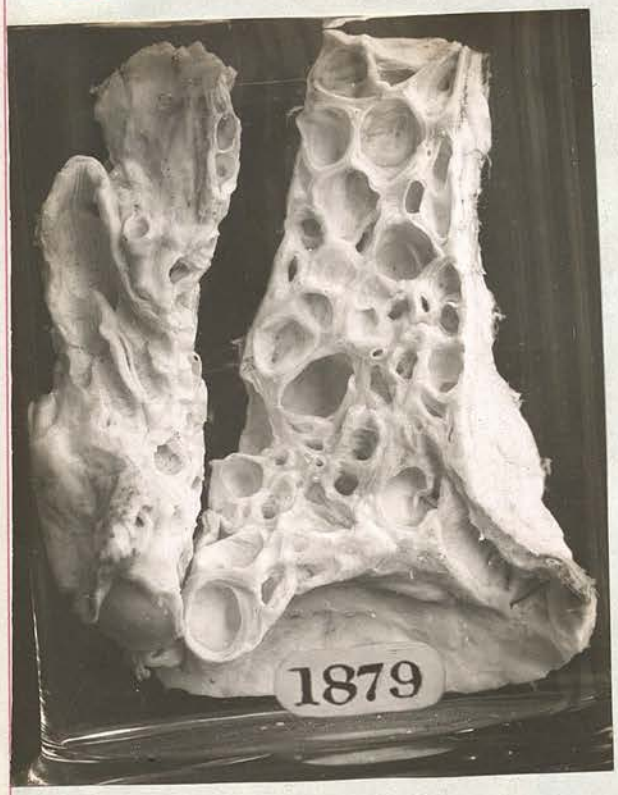
shewing a condition of almost ~~Universal~~ general Cylindrical Bronchiectasis with one or two small sacculer dilatations.

Case Peter Coleman noted in Morbid anatomy : No. 60/ duration 10½ years :

(Brompton Hospital Museum).

III :-

C



Specimen:
shewing extreme saccular Bronchiectasis in lower part of lung.
Lung tissue airless & extremely fibrotic in a male aet. 35 years.
Type: chronic Bronchitis.

(St. Thomas's Hospital museum).

IV :- XC

C

Specimen:
shewing large saccular dilatations in lower part of lung — all intercommunicating. The lung tissue is solid from Tubercular disease: but microscopic evidence the saccular cavities proves to be Bronchiectatic.

(St. Thomas's Hospital museum).

(V) :-

3361. B.

Specimen:

Left Lung :-
extreme Bronchiectasis
— sacular of
lower lobe:
Lung tissue of lower
lobe markedly
fibrotic /

In a female. aet:
22 years /

(see Right Lung
of same case
below).

(VI) :-

C

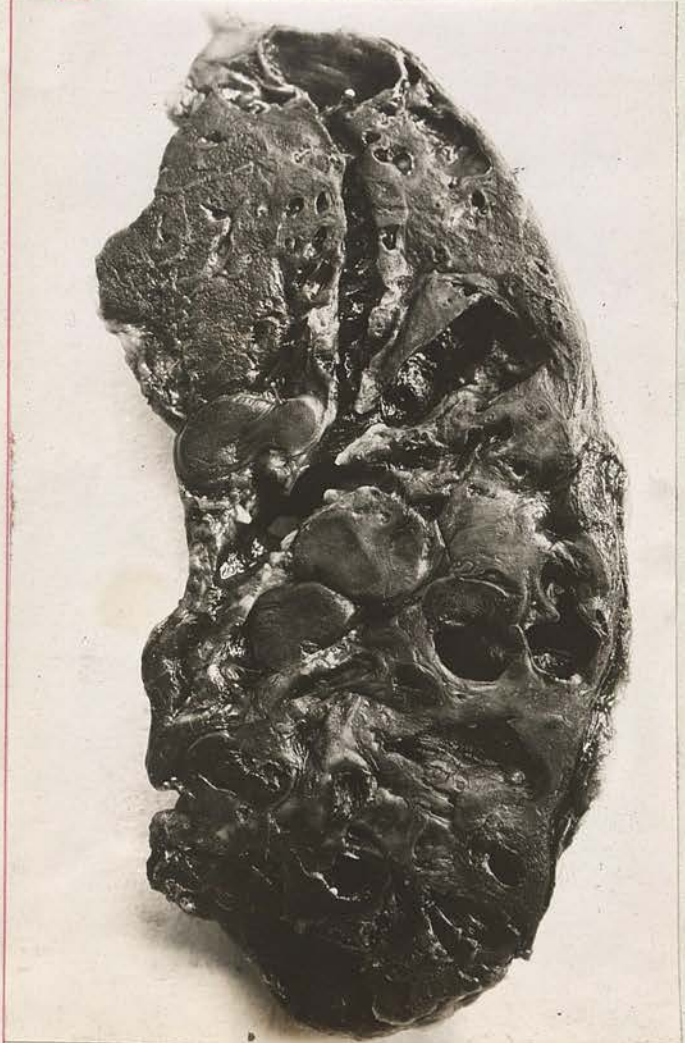
Specimen:
Right Lung :-
(of the above case).
shewing extreme
consolidation by
lobular Pneumonia.
upper two lobes
shew numerous
small cavities
resulting from
dilatation of the
bronchial tubes (?)

(Royal College of
Surgeons Museum)

Specimen :
Lung :
Shewing
marked
saccular
Bronchiectasis
& marked
cirrhosis of
lung tissue

~~Case~~ :
~~Charles Parker~~
~~aet 19~~
~~(See Morbid anatomy notes No . . .)~~

VIII :- (Brompton Hospital Museum)



Specimen :
Lung :
Shewing a General
condition of
Bronchiectasis
ie marked
saccular &
cylindrical :
Lung tissue
throughout
markedly fibrotic

(Victoria Park
Hospital for Diseases
of chest . museum)

IX :-

/.

C

Vertical section: through lung: shewing a condition of acute Bronchiolectasis note minute dilated Bronchioles. throughout. (Dr. Sharkey's case). St. Thomas's Hospital Reports Vol XXII. 1892-93. page 34.). (St. Thomas's Hospital Museum.)

note also condition of the Pleura.

X :-

C

Specimen:

External surface of the above lung showing the dilatations projecting on the surface: the elevations in the specimen are depressed - due to collapse from the spirit.

I. XI (a).

(Brompton Hospital Museum)

Specimen :-
Right Lung. -
¼ smaller than the left lung.
shewing Bronchi to lower lobe considerably dilated.
(cylindrical)
In upper & middle lobes Bronchi natural
Lung tissue around the bronchial dilatations somewhat fibrotic / associated Empyema.
Case: Emily Maclean.
(see. morbid anatomy. No. 32/
(2 other specimens of this lung to follow. below)

II XI (b) :-

(Brompton. Hospital Museum)

Specimen :
Right lung :-
shewing the marked cylindrical dilatations in lower lobe.
Case: Emily Maclean. (as above)

(Brompton Hospital Museum) XI (c) :-

Specimen :-
Right Lung:
shewing external surface of lung. ē the results of the associated Empyema /.

Case. Emily Maclean
(as above)
/

XII :-

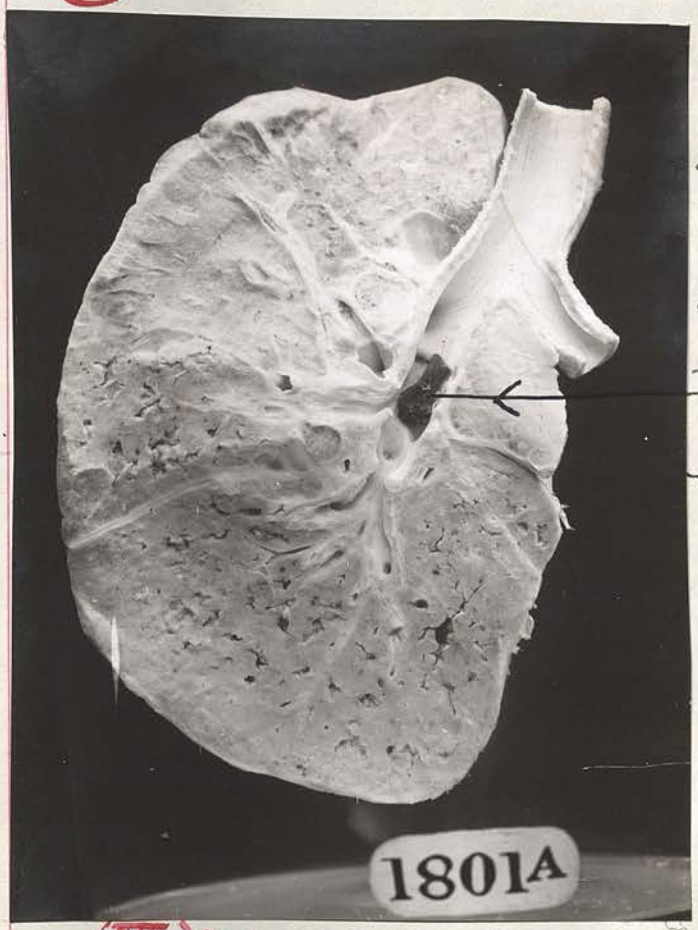
Specimen:
Right Lung:
shewing cylindrical & saccular Bronchiectasis ē large sanguenous cavity in upper & middle lobes.
just about to burst through the Pleura:

Case. John Walton. see (Morbid anatomy notes No. 29/

This is the case treated by Mr. Macgeire. by Intravenous injections of Formalin /

(Brompton Hospital Museum)

XIII :-



Specimen :-
Lung:
shewing extreme acute Broncho-Pneumonic Consolidation, (grey hepatization): riddled with abscesses.

← Clove in Bronchus

all the immediate results of impaction of a clove in the Bronchus.

Death occurred 3 weeks after the accident.
(St Thomas's Hospital Museum)

XIV :-

Specimen:
Right Lung: shewing the results of impaction of a piece of Bone in a Bronchus (main)
Collapse of lower lobe
General Bronchiectasis & some ulceration of the Bronchi.

← Piece of Bone impacted in the Bronchus

C. Pericarditis & Effusion

Case of A. J. Ceaton.
noted in Morbid Anatomy: No: 41

(Brompton Hospital Museum)

Specimen :-
Right Lung :-
shewing results of impaction of an Ear of Corn in the main Bronchus of the lung : all the Bronchi to lower lobe much dilated (cylindrical) Lung tissue (lower lobe) collapsed + airless

Ear of Corn in the Bronchus. (in situ).

middle lobe contains a cavity + bronchus communicating with it slightly dilated

Pleura over lower lobe - densely adherent.

Case : William Hodges aet. 17. (See Morbid Anat: notes No. 42)

XVI. (Brompton Hospital Museum)

Specimen :-
Larynx, Trachea, Right + left Bronchi :
History of syphilis 10 years before death.

old syphilitic scarring

Specimen : shewing Syphilitic scarring + Contraction of Trachea + Both Bronchi

Case : (See Morbid anat: notes No : 44)

Syphilitic scarring + contraction

Syphilitic Contraction

Syphilitic Contraction

Lung : slightly emphysematous cylindrical dil: slight in upper lobe : m

XVII (Brompton Hospital Museum)

Specimen :-
Right lung :-
shewing results of impaction of a tooth in a bronchus
upper & middle lobes were studded w. tubercular racemose vomicæ hard & pigmented

← Tooth impacted in a Bronchus (in situ).

at apex of lung was a small tubercular cavity

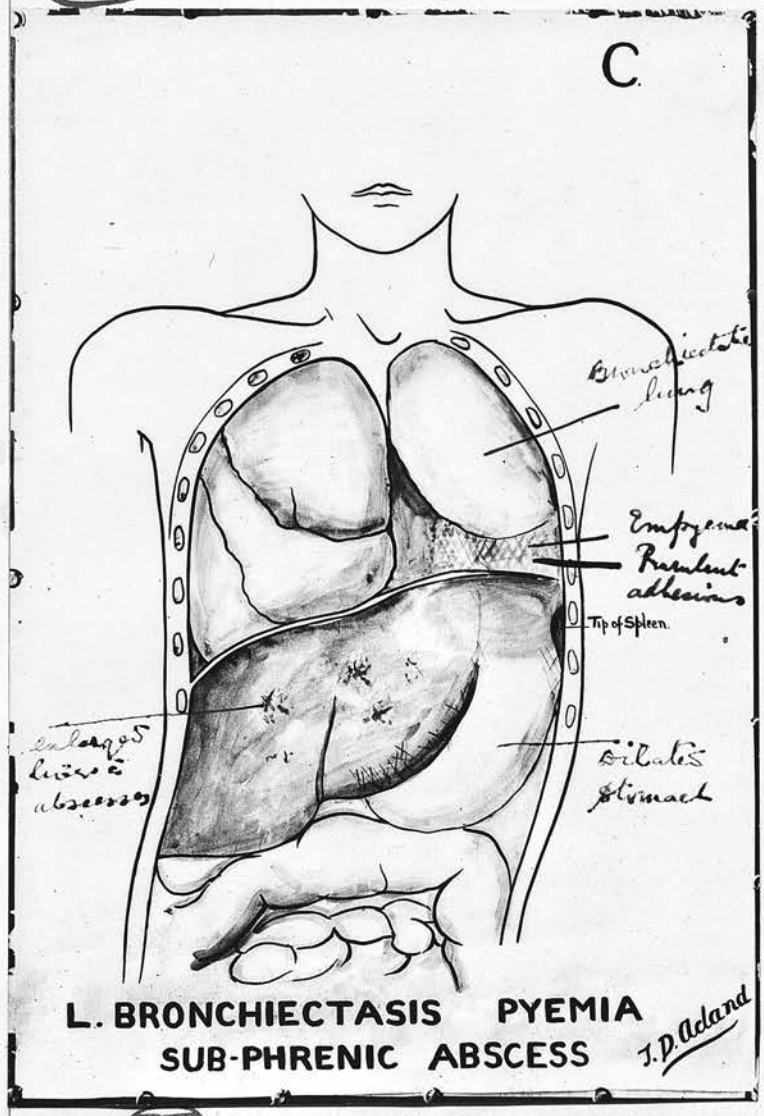
Lower lobe :-
Contracted & fibroid.
all the Bronchi thickened & dilated. here.

~~Case~~ :- ~~Emily Maclean~~
~~see morbid anatomy No~~

XVIII :-

Specimen :-
Left lung :-
shewing marked cylindrical Bronchiectasis.
Lung :
almost normal if anything a little firmer than normal
chronic Bronchitis type
Case : Charles Rowds
æt. 17
(See Morbid Anatomy Notes. No. 2.)
(Brompton Hosp. Museum)

XIX :—



Case of N. P. noted in my Morbid Anatomy notes No:

a drawing taken from the condition presented post-mortem: shewing. subdiaphragmatic abscess: acute septic Hepatitis: acute dilatation of the stomach & Empyema (left). all secondary to Bronchiectasis of left lung.

This illustration is included by the kind permission of Sir Adand Physician to St Thomas's Hospital.

XX :—

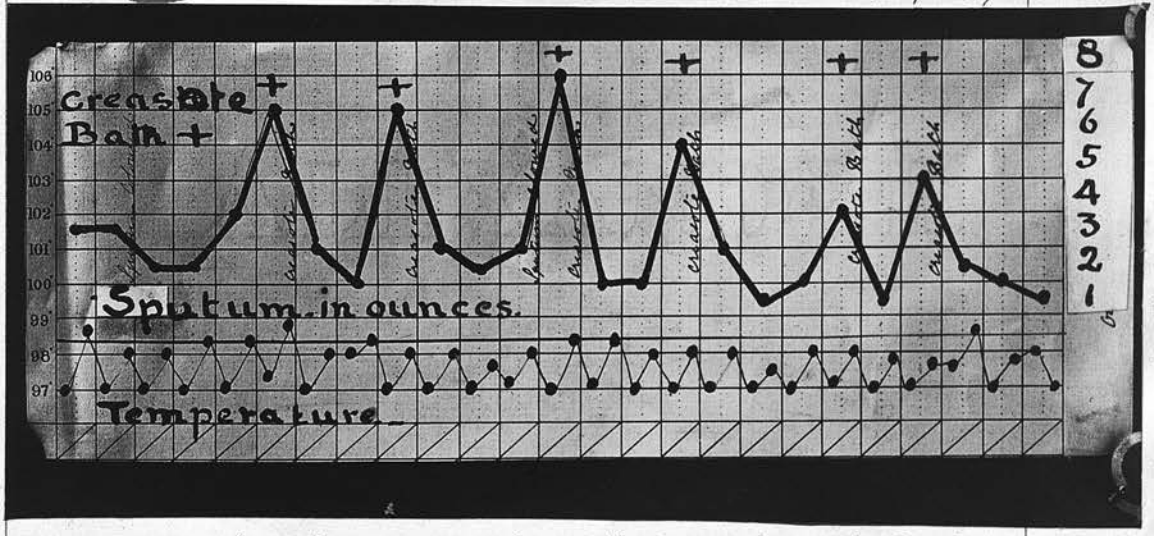
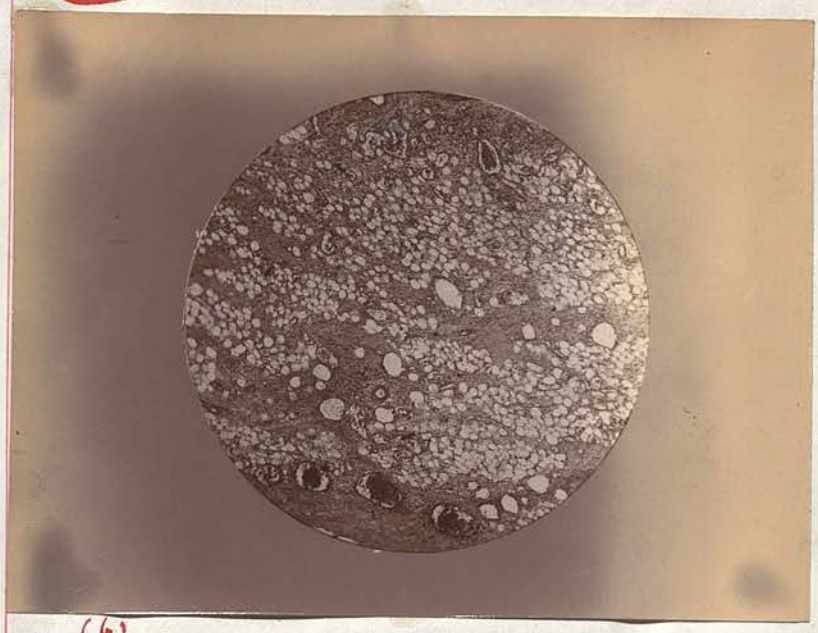


Diagram to shew immediate effect of a Creasote Bath on the expectoration:

(XX) (a+b) :- × c



(b) × c

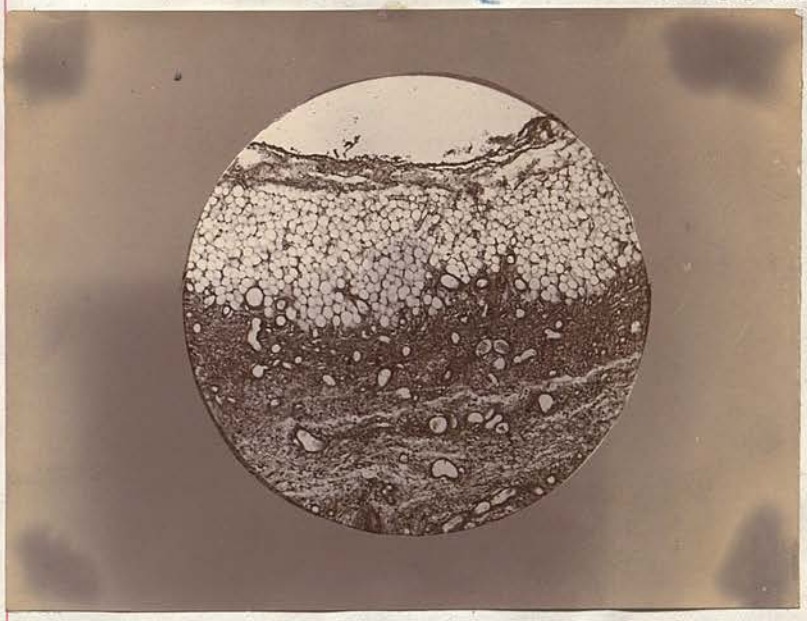
+40/

Two specimens: shewing the appearances presented in the buttock tissues, where 5·2 Guaiacol Injections were given as described in the section on Treatment. (subcutaneous). ie Case. Annie Wright. under the care of Dr. Kingston. Fowler. (The temperature chart & a few notes of the case are given at the end of my paper.) It was intended that the above specimens should be shown under the high-power. but the microphotographer unfortunately reproduced them in a low power.

The appearances under high power: shewed some sub-acute inflammatory changes. & a marked small cellular infiltration. Some haemorrhages were seen throughout. Some slight necrosis of the tissues: No abscess formation of any kind. /

XXI :-

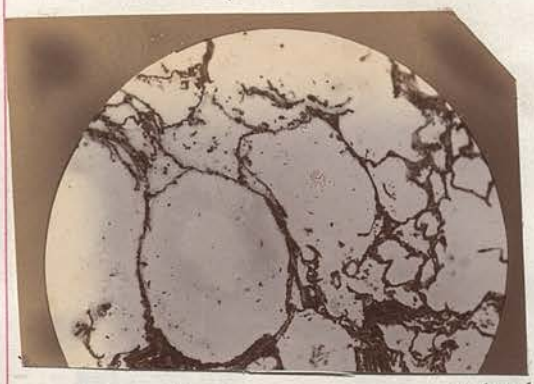
specimen :- shewing the condition of the Pleura in the left lung of a case where the right lung shewed marked Bronchiectasis. The left lung from which this specimen was taken had adherent pleura nearly all over. The lung tissue shewed distinct signs of cirrhosis + was contracting + pulling apart the layers of the pleura: This condition is well shown in the section: The Bronchial tubes were distinctly somewhat dilated.



XXII :-

specimen :- Right Lung shewing the exact similar conditions as in the above specimen - In a case where the left-lung was markedly Bronchiectatic The Right-lung was contracting under the increasing fibrosis of its tissues +, was pulling apart the adherent pleural layers. as is well-shown in the above specimen. The Bronchial tubes were somewhat distinctly dilated. + this dilatation made one incline strongly to the view, that this had certainly occurred before the (increasing) lung fibrosis (had ever occurred) /. (The cirrhosis was more marked than in the above specimen XXI.

XXIII :- X C



+40/

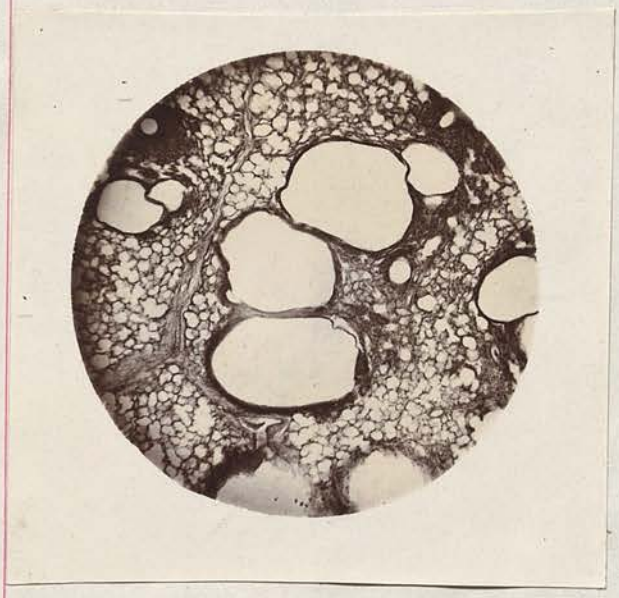
Specimen : shewing the effects of long Continued Cough (at least 15 years) on the (lung) tissue of the left lung in a case whose Right lung shewed marked Bronchiectasis. The Pleura on the left side was free. This section was taken just below the pleural surface/.

XXIV :- X C

Specimen : from left lung of A Sydney Tenner: (see X Ray. notes. Case. No VII.) The case was one of Tubercular Bronchiectasis of the Right lung : The left lung shewed diffuse racemose tubercles : & adherent pleura/. (see Morbid Anatomy Notes. A & F Case. No.)

XXV :-

X Chs.

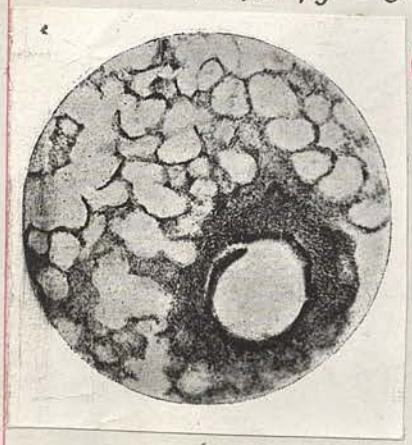


specimen :

shewing a condition of acute Bronchiolectasis the dilated Bronchioles are well shown. The lung tissue is fairly normal

This specimen was taken from a microscope specimen kindly lent by Dr Acland Physician to St Thomas's Hospital. as also is the one below)

(see St Thomas's Hospital Reports. Vol. XXII : page 1892-93 34 /.)



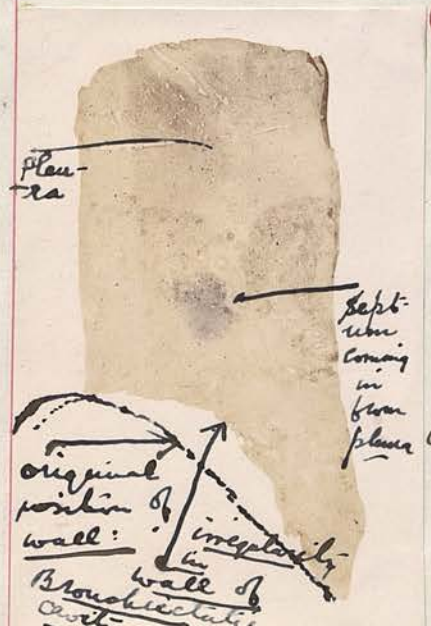
XXVI :-

specimen :-

shewing the Bronchiolar dilatation & the acute Peribronchitis & some degree of emphysema around /.

(Dr Sharkey's case)

(see Photograph of the above specimen in Photographs No. IX & No X.)



XXVII :-

specimen :

From right-lung of the case of A. Sydney Turner tuberculai Bronchiectasis (see Morbid Anatomy Notes No.). shewing as described in my paper. the contraction of the Bronchiectatic cavity causing irregularity in its outline. owing to a (thickened) septum coming in from the pleura to its walls / undoubtedly the irregularity was due to its own contraction and to the contraction of the

Morbid Anatomy :-

Explanation of Symbols used.

under Pleurae :-

+ means a moderate degree of Pleural adherency

++ means very marked Pleural adherency

— means absence of adhesions

under Bronchiectasis :-

+ means a moderate degree of Cylindrical Bronchiectasis

++ : means a very marked degree of Bronchiectasis (Cylindrical)

— means there were no Bronchiectases present-

No.	NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	Middle.	Lower.	PLEURAE. LEFT LUNG. Upper.	Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	Middle.	Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	Middle.	Lower.	LUNG TISSUE. LEFT LUNG. Upper.	Lower.
	A. S. male	40. years.	cough + expectoration as long as he can remember.	Bronchitis. Inflammation of the lung (left). 23 years ago.	General cylindrical.	Chronic Bronchitis	+	+	+	+	+	+ cylindrical small Bronchi	+ cylindrical small Bronchi	+ cylindrical small Bronchi	+ cylindrical small Bronchi	+ cylindrical small Bronchi	very emphysematous + tucky. Distinct fibrous bands passing out from the dilated bronchi into the surrounding lung tissue			very emphysematous + tucky. fibrous bands as in right lung.	
	C. R. male	17 years	5 years.	Bronchitis. Pleurisy (dry) in both sides after onset. (slight adhesions)	General cylindrical	Chronic Bronchitis	+ slight adhesions	+ slight adhesions	+ slight adhesions	+ slight adhesions	+ slight adhesions	++ marked	+	++ marked	+ slight	++ most marked	little altered firmer.	little altered firmer.	little altered firmer.	little altered firmer.	little altered firmer.
	W. Ch. male.	39 years	10 years	Bronchitis. Pneumonia 2 ago.	Slight general cylindrical + saccular.	Chronic Bronchitis	+ posteriorly only.	—	—	—	—	+ slight cylindrical & small saccular	+ slight cylindrical & a few small saccular	+ slight cylindrical & a few small saccular	+ slight cylindrical & a few small saccular	+ slight cylindrical & a few small saccular	Considerable general emphysema + congestion otherwise natural.			Considerable general emphysema otherwise natural.	
	C. A. female.	40 years	4 years.	Bronchitis.	cylindrical + saccular.	Chronic Bronchitis	++ also adhesions	++ Pericardial both sides	++ + to Diaphragm	++	++ very dense + to Diaphragm	+ cylindrical + small saccular	+ cylindrical + small saccular	+ cylindrical + small saccular	+ cylindrical + small saccular at apex + anteriorly	++ cylindrical + small saccular at base	uniform fibrous	uniform fibrous	uniform fibrous	uniform fibrous	uniform fibrous
																	scattered fibrous patches all over.			scattered fibrous patches	
	H. W. male.	35 years	3 years	Bronchitis. Pleurisy (dry) Right + left. Left	General saccular + cylindrical	Chronic Bronchitis	++	++	++ + to Diaphragm	++	++ + to Diaphragm	+ c small saccular	+ c small saccular	+ c small saccular	+ c small saccular	+ c small saccular	somewhat indurated very firm.	somewhat indurated very firm.	somewhat indurated very firm.	somewhat indurated firm.	somewhat indurated firm.
							Pleuro. Pericardial adhesions both sides.										changes more marked in this lung.				
	B. S. Female.	14½ years	4 years.	Bronchitis.	cylindrical + saccular some Fusiform.	Chronic Bronchitis.	+ spongy	++ spongy	++ spongy.	+ a few only spongy.	+ a few spongy.	++ each ending in an elongated saccule	++ each ending in an elongated saccule	++ each ending in an elongated saccule.	+ Fusiform	+ Fusiform	Chiefly emphysematous otherwise natural.			slight fibrous anterior surface emphysematous	

ME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
H.	33 years	12 months	Bronchitis — aortic stenosis.	sacculer + cylindrical	Chronic Pneumonic or Chronic Fibroïd.	++	++	++ posteriorly only	++ apex only	—	++ sacculer	++ cylindrical	+ cylindrical	+ smaller tubes very slight.	+ smaller tubes — very slight.	Highly fibroïd	Highly fibroïd	some scattered fibroïd induration	Congested otherwise natural.	
W.	38 years	2 years	Bronchitis — ague before onset	sacculer + cylindrical.	Chronic Pneumonic or Chronic Fibroïd.	—	—	—	++ + to Pericardium	++	+(?).	—	+(?).	++ ō sacculer cavity.	++ ō large sacculer cavity at upper part.	Generally emphysematous a little firmer than natural			Highly fibroïd.	Highly fibroïd.
H.	49 years	2 ?	?	one Large sacculer cavity.	Chronic Pneumonic or Chronic Fibroïd.	++ posteriorly only.	++ posteriorly only.	++ posteriorly + to diaphragm	—	—	Part of large sacculer cavity here.	—	Part of large sacculer cavity here. at the upper & back part.	—	—	Highly fibroïd	Highly fibroïd	Highly fibroïd	emphysematous — oedematous.	
N. male.	35 years	12 months	Bronchitis — Syphilis before onset.	sacculer + cylindrical	Chronic Pneumonic or Chronic Fibroïd	++	++	++	++ very firm at apex.	++	+ ō a large cavity (Granular) & some smaller cavities at Base	+	+ ō a large sacculer cavity	++ ō a large sacculer cavity & several small cavities	++ ō a large sacculer cavity & several smaller cavities	Spongy + oedematous.			dense fibroïd.	
F. L. male.	21 years	cough as long as he can remember no acute illness.	Bronchitis —	Cylindrical	Chronic Pneumonic or Chronic Fibroïd	—	+	+ anteriorly only.	++	++	—	—	—	++	++	Natural.		scattered recent Broncho-Pneumonia	Lung tissue densely fibroïd.	
E C. male.	38 years	4 months	Bronchitis — Pleurisy (?) later Syphilis.	sacculer + cylindrical.	Broncho-Pneumonic	+ recent	+	+ much firmer at apex recent	++	++	+ ō large sacculer cavities.	+ ō large sacculer cavities.	+ ō large sacculer cavities.	++ large cavity occupying part of both lobes.	+ + ō sacculer	spongy lung tissue	spongy little altered	spongy	Pale & Condensed Caseous nodules all around sacculer cavity.	

						PLEURAE.					BRONCHIECTASES.					LUNG TISSUE.				
						RIGHT LUNG.			LEFT LUNG.		RIGHT LUNG.			LEFT LUNG.		RIGHT LUNG.			LEFT LUNG.	
NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	Upper.	Middle.	Lower.	Upper.	Lower.	Upper.	Middle.	Lower.	Upper.	Lower.	Upper.	Middle.	Lower.	Upper.	Lower.
C. T. male.	29 years	Cough all his life.	Bronchitis. Pneumonia(?) (left) Later. (2 years before admission)	sacculer + cylindrical (right) some gangrene.	Broncho-Pneumonic	++	++	++	—	++	gangrenous sacculer cavity.	—	++ c large sacculer cavity c several small cavities.	—	++ c one large sacculer cavity + several small cavities	scattered Broncho-Pneumonic patches. Solid grey in colour.	?	some fibrosis Broncho-pneumonic patches post.	spongy c large pigmented nodules	much contracted densely fibrosed especially at lower part
J. L. male.	28 years	3 years	Bronchitis following Rheumatic fever.	Cylindrical + sacculer.	Broncho-Pneumonic	++ c very dense at apex especially.	++	++ c diaphragm	++	++ c diaphragm	++ c numerous small sacculer cavities.	++ c many small sacculer cavities.	++ c many small sacculer cavities.	++ c many small sacculer cavities.	++ c many small sacculer cavities.	anteriorly spongy Posteriorly consolid some cirrhosis more basalyr		anteriorly spongy Consolid more diffuse.	anteriorly spongy some fibrosis inducation	
W. S. male.	18 years	6 months.	Bronchitis + Influenza.	sacculer + cylindrical.	Broncho-Pneumonic	++	—	—	++	+ c portion basally only.	sacculer Cavity.	—	—	— large sacculer cavity.	+ c some small sacculer cavities also one large cavity	Consolidated somewhat dense	yellow areas c Broncho-Pneumonic Consolidation	solid c Broncho-Pneumonic yellow areas.	dense tough fibrosis + Cirrhosis	Congested + oedematous
A. J. male	32 years	Cough since a child.	Bronchitis	Right: Cylindrical Left: sacculer.	Broncho-Pneumonic	+ spongy but firm.	+	+	+	++ very thick.	—	—	+ small Bronchi at base only.	+(3) very slight 2 some gangrenous Cavities	riddled c large sacculer Cavities	Healthy but Congested			Broncho-Pneumonic Consolidation	some fibrosis
N. H. male.	29 3/4 years	Cough since a child.	Bronchitis Pleurisy (left) + Congestion of lungs aet. 12 years.	General Cylindrical	Broncho-Pneumonic	++	++	++	++	++	++ cylindl.	++ cylindl.	+ cylindl.	+ lower part only cylindl.	++ cylindl.	Patchy Broncho-Pneumonia some fibrosis	some fibrosis	apex like grey hepatization Base oedematous	lower part solid from Broncho-Pneumonia + firm	Broncho-Pneumonic Consolidated areas firm
M. G. male.	14 years	1 year	Bronchitis	sacculer	Broncho-Pneumonic	++	++	++	++	++	—	—	++ c one large gangrenous cavity + some smaller sacculer cavities	++ numerous sacculer small cavities	++ numerous sacculer small cavities	engorged		Base Consolidated by Broncho-Pneumonic areas.	Consolid	Consolid all in a state of Chronic Pneumonia with fibrosis

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	Middle.	Lower.	LEFT LUNG. Upper.	Lower.
9. M. B. male.	41. years	2½ years	Bronchitis.	cylindrical + saccular. — (some ulceration)	Broncho-Pneumonia.	+	+	+	+ post. surface only	+ post. surface only
10. H. D. male.	16 years	Coughs since a child.	Bronchitis + Influenza. —	Saccular & some cylindrical. —	Broncho-Pneumonia	—	—	+ + posteriorly only	+	+
11. J. L. R.	30 years	3 years.	Bronchitis & vague history of Pneumonia 2 yrs (right) later.	saccular + slight cylindrical — some Fusiform	Broncho-Pneumonia	+ few along vertebral border only	+ few along vertebral border only	+ few along vertebral border only	—	—
12. A. L. male.	6 years	Cough since birth.	Bronchitis — measles. — aet: 10 months. — ? Broncho-Pneumonia.	saccular.	Broncho-Pneumonia.	+	+	+ + to diaphragm only.	+	+ + to diaphragm only.
13. C. R. male.	40 years	15 months.	Bronchitis —	marked saccular + cylindrical (small Bronchi).	Broncho-Pneumonia & (recent septic Broncho-Pneumonia)	+ +	+ +	+ +	—	—
14. H. C. male.	43 years	5 years.	Bronchitis — smallpox. Pleurisy (right) 20 years ago.	general cylindrical	Broncho-Pneumonia	+ at upper part only	—	—	+ upper ⅓ —	+ to diaphragm only

BRONCHIECTASES.

RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
+ opening into small saccules	+ few / c some irregularly ulcerated cavities near root /	+ opening into small saccules /	+	upper ½ riddled c small saccular cavities / also at post. Base /
—	—	+ c some small saccular cavities —	—	+ c many small saccular cavities /
+ c riddled c small saccular cavities /	+ c riddled c small saccular cavities /	+ c few small saccular cavities at Base /	++ large Fusiform dilatation at Base c numerous small saccules /	
+ some saccular cavities	some saccular cavities	numerous saccular cavities.	+	numerous saccular cavities.
++ c some saccular cavities anteriorly —	? /	++ c some saccular cavities anteriorly	—	—
+ specially marked at apex /	+	+	++ most marked at apex. Base /	++ Base specially /

LUNG TISSUE.

RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
Fibrotic	emphysema Fibrosis	Fibrotic	slight induration In ant. part. tough Broncho-Pneumonic areas.	In ant. part tough Broncho-Pneumonic nodules /
Emphysematous		Patches of Broncho-Pneumonia /	some Broncho-Pneumonic areas /	more or less solid from Broncho-Pneumonia some fibrosis.
Dense: oedematous + Congested: but no obvious fibrosis /		dense + congested no obvious fibrosis	spongy	dense oedematous + congested no obvious fibrosis /
irregular patchy Consolidation soft yellowish-grey areas		Partially Collapsed some cirrhosis some Broncho-Pneumonia /	Generally spongy numerous red haemorrhagic foci of Consolidn	Partially Collapsed some cirrhosis some Broncho-pneu Consolidn
Collapsed soaked in purulent material /	?	Collapsed soaked in purulent material /	crepitant throughout almost natural /	
General reticular fibrosis Fibrosis marked at apex /	marked general reticular fibrosis /	upper ½ airless due to Broncho-pneumonia marked General reticular fibrosis	very emphysematous Considerably Collapsed & shrunken Fibrotic /	marked Broncho-Pneumonic Consolidn in lower part.

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	Middle.	Lower.	LEFT LUNG. Upper.	Lower.
5. R. H. male.	16 years	4 years.	Bronchitis	Cylindrical & gangrenous saccular Cavity/.	Broncho-Pneumonia & recent. septic Broncho-Pneumonia/	++	++	++	+	+ very slight/.
6. N. P. male.	16 years/	14 months/	Bronchitis Empyema. sub-diaphragmatic abscess. Pyaemia./.	saccular + Cylindrical & ulceration + Gangrene/	Broncho-Pneumonic	—	—	— Base has a recent layer of lymph.	++	++
7. G. L. male.	55 years.	about 4 years.	Bronchitis. Pleurisy. (right) after onset	General cylindrical + saccular	Broncho-Pneumonia	+	+ General spongy.	++ specially firm & diaphragm/	+ General Spongy	++ specially firm & diaphragm
8. J. W. male/	32 years	4 years/	Pneumonia (lobar) (Pleurisy + after onset).	Cylindrical. + saccular/.	Pneumonic. (& Broncho-Pneumonia Opens & upper lobe).	+	+ in a patchy manner.	+	++	++.
9. J. W. male/.	13 years	11 months	Pneumonia (lobar). (Typhoid set. 5 years/	marked saccular slight-cylindrical Gangrene/.	Pneumonic	+	+	+	—	—
10. J. L. male/	32 years	10 months/	Pneumonia (lobar).	marked saccular + Cylindrical	Pneumonic.	++ very dense	++ very dense	++ very dense	—	— Part & the Pericardium

BRONCHIECTASES.

RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
+ c me large Sanguenous saccular cavity/.	+	++	+ c some very small Cavities (saccular)	+(?) very slight-
a few Bronchi very slightly dilated (?).			several large saccular cavities at- apex - Sanguenous smaller at Base	+ (slight-) c several larger saccular cavities Sanguenous/.
+	+	++ c some saccular cavities/	+	++ c some saccular cavities walls ulcerated
+ slight-	+ slight-	+ slight-	++ c a few small Cavities.	riddled c small saccular cavities/
Large Sanguenous Saccular Cavity/		+ c numerous saccular cavities	—	—
++ c numerous saccular cavities	++ c numerous saccular Cavities	++ c numerous saccular Cavities	—	—

LUNG TISSUE.

RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
solid from recent- septic Broncho- Pneumonia	Patches of septic Broncho- Pneumonia	shrunken Fibrotic	Patches of septic Broncho- Pneumonia	natural/
Full of small. irregular Yellow areas of Broncho- Pneumonia			somewhat- emphysematous	Fibrotic specially so at- Base.
very deeply engorged & oedematous/ coarse fibrois reticulum. c deeply pigmented fibrois nodules		very densely fibroid/	very densely engorged & oedematous coarse fibrois reticulum fibrois nodules.	dense & generally fibroid/
some induration more or less spongy	more or less spongy/	more or less spongy/ c recent- fibrois at- Base/	slight- induration some patches of Consolidn.	Collapsed & Contracted sodden indurated
marked thickening of Interlobular septa	Interlobular septa not- thickened/		natural/	
Collapsed & oedematous c Considerable fibrois			very oedematous	

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	RIGHT LUNG. Middle.	RIGHT LUNG. Lower.	LEFT LUNG. Upper.	LEFT LUNG. Lower.
1. E. M. male.	42 years.	8 years.	Pneumonia (lobar)	General Cylindrical —	Pneumonic — c septic Broncho-Pneumonia spread	++	++	++	+ slight.	+	+	++	++	+ slight.	+	scattered septic Broncho-Pneumonia Patch of gangrene	some fibrosis — scattered septic Broncho-Pneumonia	some fibrosis — scattered septic Broncho-Pneumonia	scattered septic Broncho-Pneumonia — no patch of gangrene	
2. E. M. Female.	27 years.	9 weeks.	Pneumonia (lobar) followed by a limited Empyema.	Cylindrical /	Pneumonic. /	++ very thick anteriorly only. Emphysema lobularis.	++ thick anteriorly only.	++ posteriorly more or less free.	— only one or two light adhesions.	—	—	—	++	—	—	natural	Considerably smaller than left lung. natural	extremely fibrotic	natural.	
3. A. K. Female.	2	2	Pneumonia (lobar).	Cylindrical. /	Pneumonic. c Broncho-Pneumonia spread to left.	++	++	++	—	+ fine adhesions	++	++	++	—	++ in lower part. (recent).		collapsed & fibrosis /	extremely	equally & enormously distended Compensatory emphysema recent consolidation	
4. H. C. male /	23 years.	brought into hospital moribund. /		cylindrical	Pneumonic. ?	—	—	+	+	++ very thick over lower 2/3 /	~~—~~	~~—~~	~~—~~	~~+++~~						
											—	—	+ few	+	++	Firmer more friable than natural - otherwise natural.			Fibrotic	extremely fibrotic
5. J. P. male.	10 years.	about 6 months /	Pleurisy (double) c effusion /	saccular /	Pleuritic.	+ recent adhesions over posterior surface only. serous effusion still present /	+	+	+ recent adhesions over posterior surface serous effusion still present /	+	—	—	numerous small saccular at the Base /	numerous small saccular cavities at Base /	numerous small saccular cavities at Base /	emphysematous practically natural			greatly collapsed fibrotic /	
6. C. A. male.	53 years.	4 years.	Aortic aneurism — arterio-sclerosis /	Cylindrical (small bits).	Traumatic.	—	—	—	—	—	+	++	++ some saccular — some ulceration /	+	+	spongy — emphysema	Partially collapsed — emphysema /		emphysema some cavities partially collapsed /	emphysema spongy firm

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	PLEURAE. RIGHT LUNG. Middle.	PLEURAE. RIGHT LUNG. Lower.	PLEURAE. LEFT LUNG. Upper.	PLEURAE. LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	BRONCHIECTASES. RIGHT LUNG. Middle.	BRONCHIECTASES. RIGHT LUNG. Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	BRONCHIECTASES. LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	LUNG TISSUE. RIGHT LUNG. Middle.	LUNG TISSUE. RIGHT LUNG. Lower.	LUNG TISSUE. LEFT LUNG. Upper.	LUNG TISSUE. LEFT LUNG. Lower.
7. T. F. male.	51 years	10 years	Aortic Aneurism — Pneumonia (Right) later.	Cylindrical	Traumatic. — partly Tubercular.	++	++	++	+ apex only	+ Base only / + to Pericardium /	+	+	++	—	—	Tubercular cavity — & fibro-caseous-tubercular nodules condensed.	Condensed —	several tubercular cavities — masses cirrhosis at Base /	tubercular cavity & many fibroid tubercles	many fibroid tubercles /
8. S. J. male.	19 years /	4 months /	Mediastinal tumour (sarcoma)	Cylindrical	Traumatic.	++	++	++	—	—	++	+	++ especially at Base /	—	—	solid & Collapsed some recovering Broncho-Pneumonia			oedema intensely Congested /	
9. H. R. male.	21 years	about 3 years /	Mediastinal Tumour (sarcoma)	cylindrical & saccular /	Traumatic	++	+	++	+	+	+ few	+ few.	riddled & small saccular Cavities	—	—	airless & Collapsed		extensively fibroid /	Congested & Compressed not infiltrated by "growth"	
10. T. S. male.	35 years	9 years 6 months /	Mediastinal tumour (sarcoma) Pleuro-Pneumonia Pleurisy left at. assoc. /	saccular & Cylindrical. /	Traumatic.	+ Right /	—	—	++	++ also pleuro-pericardial /	—	—	—	+ & numerous small saccular cavities at Base /	+ & some small saccular cavities in upper 1/3 /		engorged & otherwise	oedematous healthy /	densely pigmented fibroid growth invades the Base /	growth invades apex oedematous /
11. A. J. C. male.	2	15 months	Piece of Bone in Right Bronchus (posterior division). Pleurisy (Right) twice / (Empyema)	Cylindrical.	Traumatic: — partly Tubercular /	++	— localized empyema	— recent lymph on surface	(not noted).		++	++ some ulcerated /	++	+	+	apex seat of obsolete tubercle engorged some fibrosis /	—	engorged /	entirely Collapsed solid Carnified /	engorged few obsolete fibroid tubercles	oedema
12. W. H. male.	17 years	somewhat over one year	Ear of Corn in Bronchus	cylindrical & saccular /	Traumatic.	—	++	++ & to diaphragm	—	—	—	+ saccular cavity /	++	—	~~healthy~~ —	—	some induration	Collapsed airless fibroid		Natural

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	PLEURAE. RIGHT LUNG. Middle.	PLEURAE. RIGHT LUNG. Lower.	PLEURAE. LEFT LUNG. Upper.	PLEURAE. LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	BRONCHIECTASES. RIGHT LUNG. Middle.	BRONCHIECTASES. RIGHT LUNG. Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	BRONCHIECTASES. LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	LUNG TISSUE. RIGHT LUNG. Middle.	LUNG TISSUE. RIGHT LUNG. Lower.	LUNG TISSUE. LEFT LUNG. Upper.	LUNG TISSUE. LEFT LUNG. Lower.
43. H. B. male.	46 years	2	Mediastinal Tumour (sarcoma).	Cylindrical.	Traumatic.	+ apex + posteriorly only.	—	— larger effusion.	—	—	+ slight.	+ slight.	+	—	—	occupied by tumour. Lung tissue represented by small strip at periphery.		shrunken & airless free from growth.		no gross otherwise natural.
44. W. M. male.	32 years.	12 months.	Syphilitic scarring & Contraction of Trachea & Both Bronchi.	Cylindrical.	Traumatic.	Few soft adhesions.	adhesions between diaphragm & right lobe of liver.		Few soft adhesions. Pleuro-pericardial adhesions.		+	—	—	—	—	slightly emphysematous otherwise				natural.
45. B. F. male.	29 years.	1 3/4 years.	Tuberculosis. Pleurisy (left) before onset. Pleurisy (right) after onset.	Cylindrical	Tubercular.	++	++	++	++	++	+	+	+	++	++		groups of grey tubercles.	large cavity	entirely excavated		large cavity collapsed fibrosis.
						Scar & diaphragm.			Scar & diaphragm.											
46. H. T. male.	27 years	1 3/4 years.	Pleurisy (left).	Cylindrical	Tubercular.	Few soft adhesions.			++	++ specially the Base.	+(?)	—	—	++	+		miliary tubercles.		a system of Cavities marked fibrosis.		Broncho-Pneumonic Tubercles (yellow Cicatrices)
47. J. W. male.	29 years	Completely his life.	Bronchitis. Tuberculosis.	Cylindrical	Tubercular.	+	+	+	+ apex only.	—	+	+ slight.	—	+ slight.	—		Fibro-caseous nodules.	fibro-caseous nodules Cavity	studded fibro-caseous nodules		scattered miliary tubercles.
						Pleuro-pericardial adhesions.														
48. J. J. male.	27 years	8 years	Bronchitis	Cylindrical.	Tubercular.	++ apex only & posteriorly.	+ Posterior Border only.	+	++ apex only & posterior Border.	+ posterior border only.	+ one only.	—	+ 2 only.	—	—	dense fibrosis some caseous nodules	—	some small cavities Tough.	Few fibrous nodules. old puckered cicatrices.		fairly healthy.

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	PLEURAE. RIGHT LUNG. Middle.	PLEURAE. RIGHT LUNG. Lower.	PLEURAE. LEFT LUNG. Upper.	PLEURAE. LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	BRONCHIECTASES. RIGHT LUNG. Middle.	BRONCHIECTASES. RIGHT LUNG. Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	BRONCHIECTASES. LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	LUNG TISSUE. RIGHT LUNG. Middle.	LUNG TISSUE. RIGHT LUNG. Lower.	LUNG TISSUE. LEFT LUNG. Upper.	LUNG TISSUE. LEFT LUNG. Lower.
49. E. G. Female.	45 years	12 months pleurisy (left) 3 times before mast.	Pleurisy & effusions. 6 years before onset.	Cylindrical.	Tubercular.	++	+	+	++	+	++	—	—	—	—	Densely Fibroid several cavities			Large Cavity & fibroid tubercles	Cavity & destroyed fibroid + racemose tubercles.
50. F. A. male.	45 years	about 2 years.	Bronchitis	sacculer.	Tubercular.	+ apex only.	—	—	+ apex only	—	some sacculer cavities	—	—	some sacculer cavities	—	Fibroid some small cavities /	some fibroid pass recent miliary tubercles	apex fibroid few recent miliary tubercles /	many cavities (small)	many small cavities recent tubercle emphysema
51. G. W. male.	28 years	3 years.	Bronchitis	Cylindrical	Tubercular.	++ apex only.	+ posteriorly only.	+ posteriorly only.	++ apex only.	+ posteriorly only.	←	+ small Bronchi.	+(?) small Bronchi.	+ small Bronchi.	+ small Bronchi.	+ small Bronchi.	Fibroid nodules & emphysema cavity.		small cavities /	small cavities. /
52. J. C. male.	47 years	14 years	Bronchitis	Cylindrical.	Tubercular. (Tubercular was basic in origin)	—	—	+. ± to diaphragm.	++	+	—	—	—	—	++	Numerous caseous nodules (Broncho-Pneumonic) Cavity almost-solid	almost-solid.	upper ⅓ solid Base oedema.	large Cavity caseous nodules	Cavity dense fibroid areas /
53. L. T. Female.	25 years	11 months /	Bronchitis.	Cylindrical. & ulceration /	Tubercular /	++ apex only /	—	—	—	—	++ & some ulceration /	++ one only /	+	+ ulceration /	—	Tough & Pneumonic Consolidation many cavities	Cavity —	some cavities	lower ½ Consolid-ated /	Caseous tubercle
54. W. S. male.	39 years	2 years.	Bronchitis.	Cylindrical.	Tubercular.	++	+	++ ± diaphragm /	+ (less) apex only & posteriorly	+ (less) posteriorly only /	+	—	+ slight.	—	—	Fibroid in part partially Collapsed Cavity /	Partially Consolid-ated	upper ⅓ crepitant lower ⅓ Collapsed & fibrotic	old quiescent tubercular lesion encapsuled otherwise healthy /	In lower ⅓ some Consolidated areas

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	PLEURAE. RIGHT LUNG. Middle.	PLEURAE. RIGHT LUNG. Lower.	PLEURAE. LEFT LUNG. Upper.	PLEURAE. LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	BRONCHIECTASES. RIGHT LUNG. Middle.	BRONCHIECTASES. RIGHT LUNG. Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	BRONCHIECTASES. LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	LUNG TISSUE. RIGHT LUNG. Middle.	LUNG TISSUE. RIGHT LUNG. Lower.	LUNG TISSUE. LEFT LUNG. Upper.	LUNG TISSUE. LEFT LUNG. Lower.
5. J. S. male.	?	? taken in moribund.	?	Cylindrical.	Tubercular /	+ some mid-axillary + over Base	+ scattered	+ in line	++ Universal Pleuro-Pericardial adhesions /	++	—	—	++.	—	—	Bulky 3 cavities — scanty tubercular groups /	spongy tubercular nodules	spongy Collapsed — fibroid Bands /	some fairly large cavities studded c miliary tubercles /	some Cavities studded c miliary tubercles /
6. H. B. male.	23 years	8 months.	Bronchitis — Pneumo-Thorax /	saccular + Cylindrical /	Tubercular /	++ to 3rd Rib.	Fluid. (serous)	Fluid. (serous).	+ to 2nd Rib. /	—	—	—	some saccular cavities /	+ c some saccular cavities /	many saccular cavities /	some Cavities /	some Cavities /	recent tubercles /	cavity — recent grey tubercles /	In lower half many Caseating Tubercles /
							Pleuro-Pericardial adhesions + also to diaphragm /													
7. T. F. male.	45 years /	6 years.	Bronchitis c — Pleuro-Pneumonia following /	Fusiform some saccular /	Tubercular /	++ thick	++ thick	++ thick	+ posteriorly only — recent	++ + to diaphragm thick	++ Fusiform — one saccular /	+	one saccular /	—	+	Scattered cavities a few fibroid nodules at Apex.	Fibroid	Highly fibrotic	Cone-shaped fibroid pigmented area at Apex emphysema	somewhat fibroid
8. B. M. B. male.	?	13 months /	Pneumonia (Tubercular) (?).	Saccular.	Tubercular. /	++	++	++	+ slight-	—	—	—	two saccular cavities at Base /	—	—	Converted into one large cavity tubercular /	some small cavities — some miliary tubercles /	Considerable fibroid throughout	large cavity /	many miliary tubercles /
9. E. W. Female /	4½ years	9 months.	Bronchitis — Influenza. Ecto /	Cylindrical some saccular /	Tubercular /	++ upper part- Chiefly /	+	+ apex only /	++ upper part Chiefly /	—	+ c one or two small saccular /	+ very Slight-	—	++	—	large Cavity at- apex — highly fibrotic	large cavity — highly fibrotic /	large cavity at- apex /	Converted almost entirely into a large cavity fibrotic.	large cavity, c scattered miliary Tubercles /
10. P. C. male.	33 years	10½ years.	Bronchitis. — Influenza. after Rec- Pleurisy (4/4) after work.	General Cylindrical.	Tubercular /	+	+	+	+	+	++	++	++	++	++	some a little old tubercle c some recent throughout /	degree of fibrosis oedema		Firmer than natural some fibrosis. oedema.	

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	PLEURAE. RIGHT LUNG. Middle.	PLEURAE. RIGHT LUNG. Lower.	PLEURAE. LEFT LUNG. Upper.	PLEURAE. LEFT LUNG. Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	BRONCHIECTASES. RIGHT LUNG. Middle.	BRONCHIECTASES. RIGHT LUNG. Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	BRONCHIECTASES. LEFT LUNG. Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	LUNG TISSUE. RIGHT LUNG. Middle.	LUNG TISSUE. RIGHT LUNG. Lower.	LUNG TISSUE. LEFT LUNG. Upper.	LUNG TISSUE. LEFT LUNG. Lower.
1. E. J. W. female.	23 years.	8 years.	Bronchitis.	Cylindrical	Tubercular.	?	? not described	?	?	?	—	—	—	—	+	large cavity. 5 fibro-Caseous change & small cavities in lower ½.	Crepitant- small patches of Catarrhal Pneumonia		Contracted — large Cavity — fibro-Caseous change.	scattered Miliary tubercles — some fibro-caseous nodules.
2. H. F. male.	33 years	17 months	Bronchitis — Emphysema (slight).	Cylindrical (almost general).	Tubercular	++	++ very thick	++ Emphysema (2 pints pus)	+ soft adhesions	+	+	+	+	+	+	somewhat Contracted large cavities — masses cirrhosis	large cavity — induration several small cavities	large cavity at apex. — lower part collapsed (emphysema)	some scattered tubercular nodules — no cirrhosis	many scattered tubercular nodules — no cirrhosis.
3. W. L. male.	14 years.	9 months.	Bronchitis — "Influenza" (after measles).	Cylindrical	Tubercular.	+ at apex posteriorly only	—	—	—	—	Some small saccular cavities (probably tubercular) (3)	—	—	++ ulcerated	—	entirely solid + cirrhotic	Posterior part solid + cirrhotic — few tubercular nodules	upper 3 solid + cirrhotic — few tubercular nodules.	Posterior part infiltrated + filled & elongated cavities	one or two large isolated patches of racemose tubercles — Base congested
4. T. D. P. male.	16 years.	5 months	Bronchitis	Fusiform	Tubercular	++ dense	++ dense	++ dense	+ & serous fluid 3 IV.	—	—	—	++ several large fusiform	—	—	Completely excavated.	some small cavities — a mass of racemose tubercle	some small cavities — apex infiltrated & Caseous tubercle.	large Cavity & several small ones.	apex riddled & cavities — some racemose tubercles.
5. J. H. male.	51 years	12 years	Bronchitis — Pleurisy (5 years ago most)	saccular + Cylindrical —	Tubercular	++	++ very dense.	++	+ apex only.	—	+ & a series of small saccular intercommunicating cavities.	+ one large	—	—	—	greatly Contracted pinkish Collapsed tissue	spongy	small Cavity at apex. emphysema fibro-Caseous nodules	large cavity a few fibro-Caseous nodules	cavity at apex. a few caseating nodules
6. H. R. female.	35 years	13 months.	"Inflammation of Both lungs"	saccular + Cylindrical	Tubercular.	++	++ and to Pericardium	++	++	++	++	+	++ & some saccular cavities at Base.	+	+ & some saccular cavities at Base.	One Cavity & some caseating tubercle	much caseating tubercle —	large cavity — Contracted + partially Collapsed	large cavity — shades & caseating tubercle some small cavities	Base imperfectly Collapsed — some caseous tubercles Moderate Congestion

NAME.	Age.	Duration.	Probable Cause and Associated Diseases.	Variety.	Probable Pathological Type.	PLEURAE. RIGHT LUNG. Upper.	Middle.	Lower.	PLEURAE. LEFT LUNG. Upper.	Lower.	BRONCHIECTASES. RIGHT LUNG. Upper.	Middle.	Lower.	BRONCHIECTASES. LEFT LUNG. Upper.	Lower.	LUNG TISSUE. RIGHT LUNG. Upper.	Middle.	Lower.	LUNG TISSUE. LEFT LUNG. Upper.	Lower.
7. S. H. male.	12 years	2.	2.	Cylindrical.	Tubercular.	-	—	—	++	++	—	—	—	++	—	spongy but permeated by tuberculous deposit very oedematous			large cavity highly fibroid & semicaseous deposits.	
8. C. N. male.	25 years	2	2.	Cylindrical (slight.)	Tubercular.	++	?	++ upper part only.	++	++ upper part only.	—	—	+ slight.	—	—	whole lobe almost excavated masses fibrous.	one cavity solid from racemose tubercle.	almost solid from caseating racemose tubercle.	large cavity studded & recent racemose Tubercle	upper ½ studded & recent racemose Tubercle some scattered lower down
9. A. S. F. male.	16 years	3 years.	Pneumonia (lobar) (2. tubercular) (Right.) Pleurisy left.	marked saccular & cylindrical.	Tubercular. (Pneumonia (2.) (Tuberculosis secondary (2.)).	++	++	++	++	++	+	+ middled throughout & large saccular cavities.	+	—	—	lung tissue densely Fibrotic.			scattered diffuse recent racemose tubercles.	
10. A. M. Female.	40 years	several years winter cough.	Bronchitis no acute illness.	General Bronchiolectasis.	Tubercular (Fairly normal lung tissue). (Tubercular Laryngitis.).	+ very slight at extreme apex only.	—	—	a few adhesions all over. scattered		numerous small saccules filled & muco-pus.	numerous small saccules filled & muco-pus.	numerous small saccules filled & muco-pus.	numerous small saccules filled & muco-pus.		edges of lung emphysematous lung tissue almost natural.		shews bullae.	lung somewhat emphysematous at the edges. anterior part shews some Broncho-Pneumonia.	almost natural.
1. M. B.	42 years	one year.	"Pleurisy" + "Congestion" of the lung" (2.)	saccular.	2.	++	++	++	—	—	one saccular cavity size of a walnut at the Base.	—	—	—	—	engorged & oedematous some emphysema.			engorged & oedematous some emphysema.	
2. L. V. Female.	34 years	9 years.	Bronchitis Influenza (several times). smallpox aet. 10 years.	marked cylindrical & one large saccular.		++	++	++	++	++	++	—	++ Communicating & a large saccular cavity at Base.	—	—	small & deeply engorged	partial Collapse	Considerable Collapse	small & deeply engorged otherwise healthy.	